

1,1,1-TRICHLOROETHANE

Summary

Preliminary results suggest that 1,1,1-trichloroethane (1,1,1-TCA) induces liver tumors in female mice. It was shown to be mutagenic using the Ames assay, and it causes transformation in cultured rat embryo cells. Inhalation exposure to high concentrations of 1,1,1-TCA depressed the central nervous system; affected cardiovascular function; and damaged the lungs, liver, and kidneys in animals and humans. Irritation of the skin and mucous membranes has also been associated with human exposure to 1,1,1-trichloroethane.

CAS Number: 71-55-6

Chemical Formula: CH_3CCl_3

IUPAC Name: 1,1,1-Trichloroethane

Important Synonyms and Trade Names: Methyl chloroform, chloro-
thene, 1,1,1-TCA

Chemical and Physical Properties

Molecular Weight: 133.4

Boiling Point: 74.1°C

Melting Point: -30.4°C

Specific Gravity: 1.34 at 20°C (liquid)

Solubility in Water: 480-4,400 mg/liter at 20°C (several divergent values were reported in the literature)

Solubility in Organics: Soluble in acetone, benzene, carbon tetrachloride, methanol, ether, alcohol, and chlorinated solvents

Log Octanol/Water Partition Coefficient: 2.17

Vapor Pressure: 123 mm Hg at 20°C

Vapor Density: 4.63



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Transport and Fate

1,1,1-Trichloroethane (1,1,1-TCA) disperses from surface water primarily by volatilization. Several studies have indicated that 1,1,1-trichloroethane may be adsorbed onto organic materials in the sediment, but this is probably not an important route of elimination from surface water. 1,1,1-Trichloroethane can be transported in the groundwater, but the speed of transport depends on the composition of the soil.

Photooxidation by reaction with hydroxyl radicals in the atmosphere is probably the principal fate process for this chemical.

Health Effects

1,1,1-Trichloroethane was retested for carcinogenicity because in a previous study by NCI (1977), early lethality precluded assessment of carcinogenicity. Preliminary results indicate that 1,1,1-TCA increased the incidence of combined hepatocellular carcinomas and adenomas in female mice when administered by gavage (NTP 1984). There is evidence that 1,1,1-trichloroethane is mutagenic in Salmonella typhimurium and causes transformation in cultured rat embryo cells (USEPA 1980). These data suggest that the chemical may be carcinogenic.

Other toxic effects of 1,1,1-TCA are seen only at concentrations well above those likely in an open environment. The most notable toxic effects of 1,1,1-trichloroethane in humans and animals are central nervous system depression, including anesthesia at very high concentrations and impairment of coordination, equilibrium, and judgment at lower concentrations (350 ppm and above); cardiovascular effects, including premature ventricular contractions, decreased blood pressure, and sensitization to epinephrine-induced arrhythmia; and adverse effects on the lungs, liver, and kidneys. Irritation of the skin and mucous membranes resulting from exposure to 1,1,1-trichloroethane has also been reported. The oral LD₅₀ value of 1,1,1-trichloroethane in rats is about 11,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

The acute toxicity of 1,1,1-trichloroethane to aquatic species is rather low, with the LC₅₀ concentration for the most sensitive species tested being 52.8 mg/l. No chronic toxicity studies have been done on 1,1,1-trichloroethane, but acute-chronic ratios for the other chlorinated ethanes ranged from 2.8 to 8.7. 1,1,1-Trichloroethane was only slightly bioaccumulated with a steady-state bioconcentration factor of nine and an elimination half-life of two days.

No information on the toxicity of 1,1,1-trichloroethane to terrestrial wildlife or domestic animals was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report, the lowest values of the two trichloroethanes (1,1,1 and 1,1,2) known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 18 mg/liter
Chronic toxicity: 8.4 mg/liter

Saltwater

Acute toxicity: 31.2 mg/liter
Chronic toxicity: No available data

Human Health

Criterion: 18.4 mg/liter

NIOSH Recommended Standard: 350 ppm (1,910 mg/m³)/15 min Ceiling Level

OSHA Standard: 350 ppm (1,910 mg/m³) TWA

REFERENCES

- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France. Pp. 515-531
- NATIONAL CANCER INSTITUTE (NCI). 1977. Bioassay of 1,1,1-Trichloroethane for Possible Carcinogenicity. CAS No. 71-55-6. NCI Carcinogenesis Technical Report Series No. 3. Washington, D.C. DHEW Publication No. (NIH) 77-803

- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1976. Criteria for a Recommended Standard--Occupational
Exposure to 1,1,1-Trichloroethane (Methyl Chloroform).
Washington, D.C. DHEW Publication No. (NIOSH) 76-184
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
- Data Base. Washington, D.C. October 1983
- NATIONAL TOXICOLOGY PROGRAM (NTP). 1984. Annual Plan for
Fiscal Year 1984. Research Triangle Park, N.C. DHHS
Public Health Service. NTP-84-023
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Chlorinated Ethanes. Office
of Water Regulations and Standards, Criteria and Standards
Division, Washington, D.C. October 1980. EPA 440/5-80-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for 1,1,1-Trichloroethane. Environmental
Criteria and Assessment Office, Cincinnati, Ohio. September
1984. ECAO-CIN-H005 (Final Draft)
- VERSCHUEREN, K. 1977. Handbook of Environmental Data on Organic
Chemicals. Van Nostrand Reinhold Co., New York. 659 pages
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

TETRACHLOROETHYLENE

Summary

Tetrachloroethylene (PCE, perchloroethylene) induced liver tumors when administered orally to mice and was found to be mutagenic using a microbial assay system. Reproduction toxicity was observed in pregnant rats and mice exposed to high concentrations. Animals exposed by inhalation to tetrachloroethylene exhibited liver, kidney, and central nervous system damage.

CAS Number: 127-18-4

Chemical Formula: C_2Cl_4

IUPAC Name: Tetrachloroethene

Important Synonyms and Trade Names: Perchloroethylene, PCE

Chemical and Physical Properties

Molecular Weight: 165.83

Boiling Point: 121°C

Melting Point: -22.7°C

Specific Gravity: 1.63

Solubility in Water: 150 to 200 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol, ether, and benzene

Log Octanol/Water Partition Coefficient: 2.88

Vapor Pressure: 14 mm Hg at 20°C

Transport and Fate

Tetrachloroethylene (PCE) rapidly volatilizes into the atmosphere where it reacts with hydroxyl radicals to produce HCl, CO, CO₂, and carboxylic acid. This is probably the most important transport and fate process for tetrachloroethylene in the environment. PCE will leach into the groundwater, especially in soils of low organic content. In soils with high levels of organics, PCE adsorbs to these materials and can

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be bioaccumulated to some degree. However, it is unclear if tetrachloroethylene bound to organic material can be degraded by microorganisms or must be desorbed to be destroyed. There is some evidence that higher organisms can metabolize PCE.

Health Effects

Tetrachloroethylene was found to produce liver cancer in male and female mice when administered orally by gavage (NCI 1977). Unpublished gavage studies in rats and mice performed by the National Toxicology Program (NTP) showed hepatocellular carcinomas in mice and a slight, statistically insignificant increase in a rare type of kidney tumor.¹ NTP is also conducting an inhalation carcinogenicity study. Elevated mutagenic activity was found in Salmonella strains treated with tetrachloroethylene. Delayed ossification of skull bones and sternebrae were reported in offspring of pregnant mice exposed to 2,000 mg/m³ of tetrachloroethylene for 7 hours/day on days 6-15 of gestation. Increased fetal resorptions were observed after exposure of pregnant rats to tetrachloroethylene. Renal toxicity and hepatotoxicity have been noted following chronic inhalation exposure of rats to tetrachloroethylene levels of 1,356 mg/m³. During the first 2 weeks of a subchronic inhalation study, exposure to concentrations of 1,622 ppm (10,867 mg/m³) of tetrachloroethylene produced signs of central nervous system depression, and cholinergic stimulation was observed among rabbits, monkeys, rats, and guinea pigs.

Toxicity to Wildlife and Domestic Animals

Tetrachloroethylene is the most toxic of the chloroethylenes to aquatic organisms but is only moderately toxic relative to other types of compounds. The limited acute toxicity data indicate that the LC₅₀ value for saltwater and freshwater species are similar, around 10,000 µg/liter; the trout was the most sensitive (LC₅₀ = 4,800 µg/liter). Chronic values were 840 and 450 µg/liter for freshwater and saltwater species, respectively, and an acute-chronic ratio of 19 was calculated.

No information on the toxicity of tetrachloroethylene to terrestrial wildlife or domestic animals was available in the literature reviewed.

¹J. Mennear, NTP Chemical Manager; personal communication, 1984.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

- The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic to aquatic organisms.

Freshwater

Acute toxicity: 5,280 µg/liter
Chronic toxicity: 840 µg/liter

Saltwater

Acute toxicity: 10,200 µg/liter
Chronic toxicity: 450 µg/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of tetrachloroethylene in water are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	8.0 µg/liter
10^{-6}	0.8 µg/liter
10^{-7}	0.08 µg/liter

CAG Unit Risk (USEPA): 5.1×10^{-2} (mg/kg/day)⁻¹

NIOSH Recommended Standards (air): 335 mg/m³ TWA
670 mg/m³ 15-min Ceiling Level

OSHA Standards (air): 670 mg/m³ TWA
1,340 mg/m³ Ceiling Level
2,010 mg/m³ for 5 min every 3 hr, Peak Level

REFERENCES

NATIONAL ACADEMY OF SCIENCE (NAS). 1977. Drinking Water and Health. Safe Drinking Water Committee, Washington, D.C.

NATIONAL CANCER INSTITUTE (NCI). 1977. Bioassay of Tetrachloroethylene for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 13, Washington, D.C. DHEW Publication No. (NIH) 77-813

- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Health
Assessment Document for Tetrachloroethylene (Perchloroethyl-
ene). External Review Draft No. 1, April 1979
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Tetrachloroethylene. Office
of Water Regulations and Standards, Criteria and Standards
Division, Washington, D.C. October 1980. EPA 440/5-80-073
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Tetrachloroethylene. Final Draft.
Environmental Criteria and Assessment Office, Cincinnati,
Ohio. September 1984. ECAO-CIN-HO09
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health
Assessment Document for Chloroform. Office of Health
and Environmental Assessment, Washington, D.C. September
1985. EPA 600/8-84/004F
- VERSCHUEREN, K. 1977. Handbook of Environmental Data on Organic
Chemicals. Van Nostrand Reinhold Co., New York. 659 pages
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

TRICHLOROETHYLENE

Summary

Trichloroethylene (TCE) induced hepatocellular carcinomas in mice and was mutagenic when tested using several microbial assay systems. Chronic inhalation exposure to high concentrations caused liver, kidney, and neural damage and dermatological reactions in animals.

CAS Number: 79-01-6

Chemical Formula: C_2HCl_3

IUPAC Name: Trichloroethene

Important Synonyms and Trade Names: Trichloroethene, TCE,
and ethylene trichloride

Chemical and Physical Properties

Molecular Weight: 131.5

Boiling Point: 87°C

Melting Point: -73°C

Specific Gravity: 1.4642 at 20°C

Solubility in Water: 1,000 mg/liter

Solubility in Organics: Soluble in alcohol, ether, acetone,
and chloroform

Log Octanol/Water Partition Coefficient: 2.29

Vapor Pressure: 60 mm Hg at 20°C

Vapor Density: 4.53

Transport and Fate

Trichloroethylene (TCE) rapidly volatilizes into the atmosphere where it reacts with hydroxyl radicals to produce hydrochloric acid, carbon monoxide, carbon dioxide, and carboxylic acid. This is probably the most important transport and fate process for trichloroethylene in surface water and in the upper

layer of soil. TCE adsorbs to organic materials and can be bioaccumulated to some degree. However, it is unclear whether trichloroethylene bound to organic material can be degraded by microorganisms or must be desorbed to be destroyed. There is some evidence that higher organisms can metabolize TCE. Trichloroethylene leaches into the groundwater fairly readily, and it is a common contaminant of groundwater around hazardous waste sites.

Health Effects

Trichloroethylene is carcinogenic to mice after oral administration, producing hepatocellular carcinomas (NCI 1976, NTP 1982). It was found to be mutagenic using several microbial assay systems. Trichloroethylene does not appear to cause reproductive toxicity or teratogenicity. TCE has been shown to cause renal toxicity, hepatotoxicity, neurotoxicity, and dermatological reactions in animals following chronic exposure to levels greater than 2,000 mg/m³ for 6 months. Trichloroethylene has low acute toxicity; the acute oral LD₅₀ value in several species ranged from 6,000 to 7,000 mg/kg.

Toxicity to Wildlife and Domestic Animals

There was only limited data on the toxicity of trichloroethylene to aquatic organisms. The acute toxicity to freshwater species was similar in the three species tested, with LC₅₀ values of about 50 mg/liter. No LC₅₀ values were available for saltwater species. However, a dose of 2 mg/liter caused erratic swimming and loss of equilibrium in the grass shrimp. No chronic toxicity tests were reported.

No information on the toxicity of trichloroethylene to domestic animals or terrestrial wildlife was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Toxicity

The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 45 mg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 2 mg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of trichloroethylene in water are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	27 µg/liter
10^{-6}	2.7 µg/liter
10^{-7}	0.27 µg/liter

CAG Unit Risk (USEPA): 1.1×10^{-2} (mg/kg/day)⁻¹

NIOSH Recommended Standards (air): 540 mg/m³ TWA
760 mg/m³ 10-min Ceiling Level

OSHA Standards (air): 540 mg/m³ TWA
1,075 mg/m³/15-min Ceiling Level
1,620 mg/m³ for 5 min every 3 hr,
Peak Concentration

REFERENCES

- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France. Pp. 545-572
- NATIONAL CANCER INSTITUTE (NCI). 1976. Bioassay of Trichloroethylene for Possible Carcinogenicity. CAS No. 79-01-6. NCI Carcinogenesis Technical Report Series No. 2, Washington, D.C. DHEW Publication No. (NIH) 76-802
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Carcinogenesis Bioassay of Trichloroethylene. CAS No. 79-01-6. NTP 81-84, NIH Publication No. 82-1799
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

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U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Trichloroethylene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 400/5-80-077

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1983. Health Assessment Document for Trichloroethylene. Review Draft. Washington, D.C. EPA 600/8-82-0068

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Trichloroethylene. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO09

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health Assessment Document for Chloroform. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA 600/8-84/004F

VERSCHUEREN, K. 1977. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York. 659 pages

WATERS, E.M., GERSTNER, H.B., and HUFF, J.E. 1977. Trichloroethylene: 1. An overview. J. Toxicol. Environ. Health 2:674-700

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

1,1-DICHLOROETHYLENE

Summary

1,1-Dichloroethylene (VDC, vinylidene chloride) caused kidney tumors (in males only) and leukemia in one study of mice exposed by inhalation, but the results of other studies were equivocal or negative. 1,1-Dichloroethylene is mutagenic, and it caused adverse reproductive effects when administered to rats and rabbits by inhalation. Chronic exposure causes liver damage, and acute exposure to high doses produces nervous system damage.

CAS Number: 75-35-4

Chemical Formula: CH_2CCl_2

IUPAC Name: 1,1-Dichloroethene

Important Synonyms and Trade Names: Vinylidene chloride, VDC,
1,1-dichloroethene, 1,1-DCE

Chemical and Physical Properties

Atomic Weight: 96.94

Boiling Point: 37°C

Melting Point: -122.1°C

Specific Gravity: 1.218 at 20°C

Solubility in Water: 400 mg/liter at 20°C

Solubility in Organics: Sparingly soluble in alcohol, ether,
acetone, benzene, and chloroform

Log Octanol/Water Partition Coefficient: 1.48

Vapor Pressure: 500 mm Hg at 20°C

Vapor Density: 3.25

Transport and Fate

Volatilization appears to be the primary transport process for 1,1-dichloroethylene (VDC), and its subsequent photooxida-

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tion in the atmosphere by reaction with hydroxyl radicals is apparently the predominant fate process. Information on other transport and fate mechanisms was generally lacking for 1,1-dichloroethylene. However, by inference from related compounds, hydrolysis, sorption, bioaccumulation, biotransformation, and biodegradation probably all occur but at rates too slow to be of much significance.

Health Effects

1,1-Dichloroethylene caused kidney tumors in males and leukemia in males and females in one study of mice exposed by inhalation, gave equivocal results in other inhalation studies, and gave negative results in rats and mice following oral exposure and in hamsters following inhalation exposure. VDC was mutagenic in several bacterial assays. 1,1-Dichloroethylene did not appear to be teratogenic but did cause embryotoxicity and fetotoxicity when administered to rats and rabbits by inhalation. Chronic exposure to oral doses of VDC as low as 5 mg/kg/day caused liver changes in rats. Acute exposure to high doses causes central nervous system depression, but neurotoxicity has not been associated with low-level chronic exposure. The oral LD₅₀ value for the rat is 1,500 mg/kg, and for the mouse it is 200 mg/kg.

Toxicity to Wildlife and Domestic Animals

1,1-Dichloroethylene is not very toxic to freshwater or saltwater species, with acute LC₅₀ values generally ranging from 80 to 200 mg/liter. A chronic study in which no adverse effects were observed indicated that the acute-chronic ratio was less than 40; a 13-day study that produced an LC₅₀ of 29 mg/liter indicated that the acute-chronic ratio is greater than 4.

No reports of the toxicity of 1,1-dichloroethylene to terrestrial wildlife or domestic animals were found in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are inadequate for establishing criteria. However, EPA did report the lowest values known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 11,600 µg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 224,000 µg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of 1,1-dichloroethylene in water are:

<u>Risk</u>	<u>Concentration</u>
10 ⁻⁵	0.33 µg/liter
10 ⁻⁶	0.033 µg/liter
10 ⁻⁷	0.0033 µg/liter

CAG Unit Risk (USEPA): 1.16 (mg/kg/day)⁻¹

REFERENCES

- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol. 19: Some Monomers, Plastics and Synthetic Elastomers, and Acrolein. World Health Organization, Lyon, France
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Carcinogenesis Bioassay of Vinylidene Chloride (CAS No. 75-35-4) in F344 Rats and B6C3F₁ Mice (Gavage Study). NTP Technical Report Series No. 228. Washington, D.C. DHHS Publication No. (NIH) 82-1784
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Dichloroethylenes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-041

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for 1,1-Dichloroethylene. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO51

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health Assessment Document for Chloroform. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA 600/8-84/004F

VERSCHUEREN, K. 1977. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York. 659 pages

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

TRICHLOROFLUOROMETHANE

Summary

Inhalation exposure to high concentrations of trichlorofluoromethane adversely affects the heart and lungs in humans and animals.

CAS Number: 75-69-4

Chemical Formula: CCl_3F

IUPAC Name: Fluorotrichloromethane

Important Synonyms and Trade Names: Freon-11, fluorocarbon 11

Chemical and Physical Properties

Molecular Weight: 137.37

Boiling Point: 23.82 °C

Melting Point: -111 °C

Specific Gravity: 1.467 at 25 °C

Solubility in Water: 1,100 mg/liter

Solubility in Organics: Soluble in alcohol, ether, and other organic solvents

Log Octanol/Water Partition Coefficient: 2.53

Vapor Pressure: 667.4 mm Hg at 20 °C

Vapor Density: 5.04

Transport and Fate

Though no specific data are available, the high vapor pressure, low solubility, and low boiling point of trichlorofluoromethane make it likely that volatilization into the atmosphere is the major transport process for removal of this compound from aqueous systems. Once in the troposphere, trichlorofluoromethane remains stable and eventually diffuses upward to the stratosphere or is carried back to earth by precipitation.

Trichlorofluoromethane that reaches the stratosphere is broken down by high energy, short wavelength ultraviolet light and this process is thought to be its primary environmental fate. Chlorine atoms released by such photodissociation processes are theorized by some researchers to serve as a catalyst in destruction of the stratospheric ozone layer.

Photolysis, oxidation, and hydrolysis do not appear to be significant environmental fate processes for trichlorofluoromethane in aquatic systems. The log octanol/water partition coefficient of trichlorofluoromethane indicates that adsorption onto sediments may occur. However, data concerning sorption processes are inconclusive.

Health Effects

Based on limited available information, trichlorofluoromethane does not appear to be carcinogenic in animals or humans. Results of a National Cancer Institute Carcinogenesis Bioassay using mice were negative. However, results for rats were considered inconclusive because inadequate numbers of rats survived long enough to be at risk from late-developing tumors. Although genotoxicity data are scant, trichlorofluoromethane exhibits no mutagenic activity in Salmonella tester strains. There are no available data on the teratogenicity or reproductive toxicity of trichlorofluoromethane.

In humans, trichlorofluoromethane toxicity generally involves the intentional or unintentional acute inhalation of high vapor concentrations. There are reports of severe intoxication and death under such circumstances. The cardiovascular and bronchopulmonary actions of trichlorofluoromethane are its two most important toxicological features and are thought to be mediated at least in part by metabolic products that bind to lipid and protein cell constituents and affect vital processes such as cellular oxidation.

The LC₅₀ value for a 4-hour exposure with rats is 26,200 ppm. During exposure, sublethal doses caused rapid respiration with some mild hyperactivity, while lethal doses caused hyperactivity, tremors, inactivity, irregular respiration, and death within four hours. Laboratory animals periodically exposed at high concentrations for several days may exhibit biochemical changes consistent with slowing of cellular oxidation. Furthermore, studies with experimental animals suggest that inhalation exposure to high concentrations of trichlorofluoromethane may produce various cardiovascular and circulatory abnormalities. Both absorption and elimination are relatively rapid in humans and experimental animals.

Toxicity to Wildlife and Domestic Animals

Data concerning the toxicity of trichlorofluoromethane to wildlife and domestic animals are not available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Criterion: 32.3 mg/liter (for protection against the noncarcinogenic effects of trichlorofluoromethane in ambient water)

OSHA Standard: 1,000 ppm (5,600 mg/m³) Ceiling Level

ACGIH Threshold Limit Value: 1,000 ppm (5,600 mg/m³) Ceiling Level

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Trichlorofluoromethane for Possible Carcinogenicity. (CAS No. 75-69-4) NCI Carcinogenesis Technical Report Series No. 106. DHEW Publication No. (NIH) 78-1356
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials. 4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Halomethanes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-051

CHLOROFORM

Summary

Chloroform (trichloromethane) is often produced during the chlorination of drinking water and thus is a common drinking water contaminant. It is volatile in surface waters and is not likely to be persistent in the environment. Chloroform caused an increase in kidney epithelial tumors in rats and in hepatocellular carcinomas in mice. In addition, there is suggestive evidence from epidemiological studies that exposure to chloroform and other trihalomethanes is associated with an increased incidence of bladder tumors in humans. Other toxic effects of chloroform include central nervous system depression; eye, skin, and gastrointestinal irritation; and damage to the liver, heart, and kidney.

CAS Number: 67-66-3

Chemical Formula: CHCl_3

IUPAC Name: Trichloromethane

Chemical and Physical Properties

Molecular Weight: 119.38

Boiling Point: 61.7°C

Melting Point: -63.5°C

Specific Gravity: 1.4832 at 20°C

Solubility in Water: 8,200 mg/liter at 20°C

Solubility in Organics: Soluble in acetone; miscible with alcohol, ether, benzene, and ligroin

Log Octanol/Water Partition Coefficient: 1.97

Vapor Pressure: 150.5 mm Hg at 20°C

Vapor Density: 4.12

Transport and Fate

Volatilization into the atmosphere is the major transport process for removal of chloroform from aquatic systems (USEPA 1979). Once in the troposphere, chloroform is attacked by hydroxyl radicals with the subsequent formation of phosgene (COCl_2) and possibly chlorine oxide (ClO) radicals. Neither of these reaction products is likely to persist; phosgene is readily hydrolyzed to hydrochloric acid and carbon dioxide. Reaction with hydroxy radicals is thought to be the primary environmental fate of chloroform. However, chloroform that remains in the troposphere may return to earth in precipitation or adsorbed on particulates, and a small amount may diffuse upward to the stratosphere where it photodissociates via interaction with ultraviolet light.

Photolysis, hydrolysis, and sorption do not appear to be significant environmental fate processes for chloroform. However, sorption processes may have some importance as a removal mechanism in groundwater and soil. The log octanol/water partition coefficient indicates that this compound may bioaccumulate under conditions of constant exposure. Studies with marine organisms provide evidence for only weak to moderate bioaccumulation. Although chloroform is somewhat lipophilic and tends to be found at higher concentrations in fatty tissues, there is no evidence for biomagnification in aquatic food chains.

Health Effects

Chronic administration of chloroform by gavage is reported to produce a dose-related increase in the incidence of kidney epithelial tumors in rats and a dose-related increase in the incidence of hepatocellular carcinomas in mice (IARC 1979, USEPA 1980). Epidemiological studies suggest that higher concentrations of chloroform and other trihalomethanes in water supplies may be associated with an increased frequency of bladder cancer in humans. However, these results are not sufficient to establish causality. An increased incidence of fetal abnormalities was reported in offspring of pregnant rats exposed to chloroform by inhalation. Oral doses of chloroform that caused maternal toxicity produced relatively mild fetal toxicity in the form of reduced birth weights. There are limited data suggesting that chloroform has mutagenic activity in some test systems. However, negative results have been reported for bacterial mutagenesis assays.

Humans may be exposed to chloroform by inhalation, ingestion, or skin contact. Toxic effects include local irritation of the skin or eyes, central nervous system depression, gastrointestinal irritation, liver and kidney damage, cardiac arrhythmia, ventricular tachycardia, and bradycardia. Death from

chloroform overdosing can occur and is attributed to ventricular fibrillation. Chloroform anesthesia can produce delayed death as a result of liver necrosis.

Exposure to chloroform by inhalation, intragastric administration, or intraperitoneal injection produces liver and kidney damage in laboratory animals. The oral LD₅₀ and inhalation LC₅₀ values for the rat are 908 mg/kg and 39,000 mg/m³ per 4 hours, respectively (ACGIH 1980).

Toxicity to Wildlife and Domestic Animals

Limited information is available concerning the toxicity of chloroform to organisms exposed at known concentrations (USEPA 1980). Median effect concentrations for two freshwater and one invertebrate species range from 28,900 to 115,000 µg/liter. Twenty-seven day LC₅₀ values of 2,030 and 1,240 µg/liter were reported for embryo-larval tests with rainbow trout in water at two levels of hardness. The only reliable result concerning the toxicity of chloroform to saltwater aquatic life is a 96-hour LC₅₀ value of 81,500 µg/liter for pink shrimp.

An equilibrium bioconcentration factor of six with a tissue half-life of less than 1 day was determined for the bluegill. Although chloroform is not strongly bioaccumulated, it is thought to be widely distributed in the environment and can be detected in fish, water birds, marine mammals, and various crops.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of chloroform in water are:

<u>Risk</u>	<u>Concentration</u>
10 ⁻⁵	1.90 µg/liter
10 ⁻⁶	0.19 µg/liter
10 ⁻⁷	0.019 µg/liter

CAG Unit Risk (USEPA): $8.1 \times 10^{-2} (\text{mg/kg/day})^{-1}$

Primary Drinking Water Standard: 0.10 mg/liter (total trihalo-
methanes)

NIOSH Recommended Standard: 9.8 mg/m³ 1-hr Ceiling Level

OSHA Standard: 244 mg/m³ Ceiling Level

ACGIH Threshold Limit Value: 50 mg/m³ (suspected human
carcinogen)

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).
1980. Documentation of the Threshold Limit Values. 4th
ed. Cincinnati, Ohio. 488 pages
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979.
IARC Monographs on the Evaluation of Carcinogenic Risk
of Chemicals to Humans. Vol. 20: Some Halogenated Hydro-
carbons. World Health Organization, Lyon, France. Pp. 408-415
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials.
4th ed. Van Nostrand Reinhold Co., New York
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Chloroform. Office of Water
Regulations and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-033
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Chloroform. Environmental Criteria
and Assessment Office, Cincinnati, Ohio. September 1984.
ECAO-CIN-HO10 (Final Draft)
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health
Assessment Document for Chloroform. Office of Health
and Environmental Assessment, Washington, D.C. September
1985. EPA 600/8-84/004F
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2332 pages

BENZENE

Summary

Benzene is an important industrial solvent and chemical intermediate. It is rather volatile, and atmospheric photooxidation is probably an important fate process. Benzene is a known human carcinogen, causing leukemia in exposed individuals. It also adversely affects the hematopoietic system. Benzene has been shown to be fetotoxic and to cause embryolethality in experimental animals. Exposure to high concentrations of benzene in the air causes central nervous system depression and cardiovascular effects, and dermal exposure may cause dermatitis.

CAS Number: 71-43-2

IUPAC Name: Benzene

Chemical Formula: C_6H_6

Chemical and Physical Properties

Molecular Weight: 78.12

Boiling Point: 80.1°C

Melting Point: 5.56°C

Specific Gravity: 0.879 at 20°C

Solubility in Water: 1,780 mg/liter at 25°C

Solubility in Organics: Miscible with ethanol, ether, acetic acid, acetone, chloroform, carbon disulfide, and carbon tetrachloride

Log Octanol/Water Partition Coefficient: 1.95-2.13

Vapor Pressure: 75 mm Hg at 20°C

Vapor Density: 2.77

Flash Point: -11.1°C

Transport and Fate

Volatilization appears to be the major transport process of benzene from surface waters to the ambient air, and atmospheric transport of benzene occurs readily (USEPA 1979). Although direct oxidation of benzene in environmental waters is unlikely, cloud chamber data indicate that it may be photo-oxidized rapidly in the atmosphere. Inasmuch as volatilization is likely to be the main transport process accounting for the removal of benzene from water, the atmospheric destruction of benzene is probably the most likely fate process. Values for benzene's log octanol/water partition coefficient indicate that adsorption onto organic material may be significant under conditions of constant exposure. Sorption processes are likely removal mechanisms in both surface water and groundwater. Although the bioaccumulation potential for benzene appears to be low, gradual biodegradation by a variety of microorganisms probably occurs. The rate of benzene biodegradation may be enhanced by the presence of other hydrocarbons.

Health Effects

Benzene is a recognized human carcinogen (IARC 1982). Several epidemiological studies provide sufficient evidence of a causal relationship between benzene exposure and leukemia in humans. Benzene is a known inducer of aplastic anemia in humans, with a latent period of up to 10 years. It produces leukopenia and thrombocytopenia, which may progress to pancytopenia. Similar adverse effects on the blood-cell-producing system occur in animals exposed to benzene. In both humans and animals, benzene exposure is associated with chromosomal damage, although it is not mutagenic in microorganisms. Benzene was fetotoxic and caused embryoletality in experimental animals.

Exposure to very high concentrations of benzene [about 20,000 ppm (66,000 mg/m³) in air] can be fatal within minutes (IARC 1982). The prominent signs are central nervous system depression and convulsions, with death usually following as a consequence of cardiovascular collapse. Milder exposures can produce vertigo, drowsiness, headache, nausea, and eventually unconsciousness if exposure continues. Deaths from cardiac sensitization and cardiac arrhythmias have also been reported after exposure to unknown concentrations. Although most benzene hazards are associated with inhalation exposure, dermal absorption of liquid benzene may occur, and prolonged or repeated skin contact may produce blistering, erythema, and a dry, scaly dermatitis.

Toxicity to Wildlife and Domestic Animals

The EC₅₀ values for benzene in a variety of invertebrate and vertebrate freshwater aquatic species range from 5,300 µg/liter to 386,000 µg/liter (USEPA 1980). However, only values for the rainbow trout (5,300 µg/liter) were obtained from a flow through test and were based on measured concentrations. Results based on unmeasured concentrations in static tests are likely to underestimate toxicity for relatively volatile compounds like benzene. A chronic test with Daphnia magna was incomplete, with no adverse effects observed at test concentrations as high as 98,000 µg/liter.

For saltwater species, acute values for one fish and five invertebrate species range from 10,900 µg/liter to 924,000 µg/liter. Freshwater and saltwater plant species that have been studied exhibit toxic effects at benzene concentrations ranging from 20,000 µg/liter to 525,000 µg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest concentrations of benzene known to cause toxic effects in aquatic organisms.

Freshwater

Acute toxicity: 5,300 µg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 5,100 µg/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of benzene in water are:

<u>Risk</u>	<u>Concentration</u>
10 ⁻⁵	6.6 µg/liter
10 ⁻⁶	0.66 µg/liter
10 ⁻⁷	0.066 µg/liter

CAG Unit Risk (USEPA): 2.9×10^{-2} (mg/kg/day)⁻¹

OSHA Standards: 30 mg/m³ TWA
75 mg/m³ Ceiling Level
150 mg/m³ 10-min Peak Level

ACGIH Threshold Limit Values: Suspected human carcinogen
30 mg/m³ TWA
75 mg/m³ STEL

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- BRIEF, R.S., LYNCH, J., BERNATH, T., and SCALA, R.A. 1980. Benzene in the workplace. Am. Ind. Hyg. Assoc. J. 41:616-623
- DEAN, B.J. 1978. Genetic toxicology of benzene, toluene, xylenes, and phenols. Mutat. Res. 47:75-97
- HAAK, H.L. 1980. Experimental drug-induced aplastic anemia. Clin. Hematol. 9:621-639
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1974. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol. 7: Some Anti-Thyroid and Related Substances, Nitrofurans, and Industrial Chemicals. World Health Organization, Lyon, France
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. An evaluation of chemicals and industrial processes associated with cancer in humans based on human and animal data. IARC Monographs Volumes 1 to 20. Cancer Res. 40:1-12
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 29: Some Industrial Chemicals and Dyestuffs. World Health Organization, Lyon, France
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Benzene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-018

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Benzene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO37 (Final Draft)

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health Assessment Document for Chloroform. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA 600/8-84/004F

WALDRON, H.A. 1979. Target organs: The blood. J. Soc. Occup. Med. 29:65-71

PHENANTHRENE

Summary

Phenanthrene is a polycyclic aromatic hydrocarbon (PAH) and is moderately persistent in natural environments. In two skin painting studies, it produced application-site tumors, and it was shown to be mutagenic in several other studies. Workers exposed to materials containing phenanthrene developed chronic dermatitis and other skin disorders.

CAS Number: 85-01-8

Chemical Formula: $C_{14}H_{10}$

IUPAC Name: Phenanthrene

Chemical and Physical Properties

Molecular Weight: 178.24

Boiling Point: 340°C

Melting Point: 101°C

Specific Gravity: 1.025

Solubility in Water: 1.29 mg/liter at 25°C

Solubility in Organics: Soluble in alcohol, ether, acetone, benzene, and acetic acid

Log Octanol/Water Partition Coefficient: 4.46

Vapor Pressure: 6.8×10^{-4} mm Hg at 20°C

Vapor Density: 6.14

Transport and Fate

Much of the information concerning transport and fate is inferred from data for polycyclic aromatic hydrocarbons (PAHs) in general because specific information for phenanthrene is lacking. Rapid, direct photolysis of phenanthrene to quinones may occur in aqueous solution. Oxidation is probably too slow to be a significant environmental process and the available data suggest that volatilization generally is not an important transport process. The calculated log octanol/water partition

coefficient of 4.46 indicates that the compound should be strongly absorbed onto particulate matter, especially particulates high in organic content. It is likely that phenanthrene can be transported as absorbed matter on suspended particulates in air or water. Data for PAHs in general indicate that phenanthrene will accumulate in the sediment and biota of the aquatic environment. Removal rates associated with absorption and subsequent sedimentation are probably slower than photolysis and degradation, but may be competitive with volatilization.

Data for a variety of PAHs suggest that bioaccumulation is a short term process, and long-term partitioning into biota is not a significant fate process. Phenanthrene can be metabolized by multicellular organisms and degraded by microbes.

Degradation by mammals is likely to be incomplete, with parent compound and the metabolites being excreted by the urinary system. Biodegradation by microorganisms is probably the ultimate fate process. Biodegradation generally appears to be more efficient in soil than in aquatic systems. However, it may be more important in those aquatic systems which are chronically affected by PAH contamination. Phenanthrene is stable enough in air to be transported over relatively great distances.

Health Effects

There are no epidemiological or case studies available suggesting that phenanthrene is carcinogenic in humans. This compound generally is not considered to be carcinogenic in experimental animals. However, at least two skin painting studies report development of tumors at the site of application in mice. Phenanthrene exhibits mutagenic activity in some test systems, but not in others. There are no reports of teratogenic or reproductive effects due to phenanthrene exposure.

Little information concerning acute and chronic toxic effects is available. Although specific data concerning exposure to phenanthrene are not available, workers exposed to materials containing this compound may exhibit chronic dermatitis, hyperkeratoses, and other skin disorders.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of toxicity to domestic animals and wildlife are not available. A 96-hour LC_{50} value of 600 $\mu\text{g/liter}$ is reported for a saltwater polychaete worm exposed to a crude oil fraction containing phenanthrene. The weighted average bioconcentration factor for the edible portion of all freshwater and estuarine aquatic organisms consumed by Americans is 486.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of carcinogenic PAHs in water are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	28 ng/liter
10^{-6}	2.8 ng/liter
10^{-7}	0.28 ng/liter

REFERENCES

- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1984. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. April 1984
- SANTODONATO, J., HOWARD, P., and BASU, D. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. J. Environ. Path. and Toxicol. 5:1-364
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials. 4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-069
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Phenanthrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H029 (Final Draft)
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2332 pages

TOLUENE

Summary

Toluene has been shown to be embryotoxic in experimental animals, and the incidence of cleft palate increased in the offspring of dosed mice. Chronic inhalation exposure to high levels of toluene caused cerebellar degeneration and an irreversible encephalopathy in animals. In humans, acute exposure depressed the central nervous system and caused narcosis.

CAS Number: 108-88-3

Chemical Formula: $C_6H_5CH_3$

IUPAC Name: Methylbenzene

Important Synonyms and Trade Names: Toluol, phenylmethane

Chemical and Physical Properties

Molecular Weight: 92.13

Boiling Point: 110.6°C

Melting Point: -95°C

Specific Gravity: 0.8669 at 20°C

Solubility in Water: 534.8 mg/liter

Solubility in Organics: Soluble in acetone, ligroin, and carbon disulfide; miscible with alcohol, ether, benzene, chloroform, glacial acetic acid, and other organic solvents

Log Octanol/Water Partition Coefficient: 2.69

Vapor Pressure: 28.7 mm Hg at 25°C

Vapor Density: 3.14

Flash Point: 4.4°C

Transport and Fate

Volatilization appears to be the major route of removal of toluene from aquatic environments, and atmospheric reactions of toluene probably subordinate all other fate processes (USEPA 1979). Photooxidation is the primary atmospheric fate process for toluene, and benzaldehyde is reported to be the principal organic product. Subsequent precipitation or dry deposition can deposit toluene and its oxidation products into aquatic and terrestrial systems. Direct photolytic cleavage of toluene is energetically improbable in the troposphere, and oxidation and hydrolysis are probably not important as aquatic fates.

The log octanol/water partition coefficient of toluene indicates that sorption processes may be significant. However, no specific environmental sorption studies are available, and the extent to which adsorption by sedimentary and suspended organic material may interfere with volatilization is unknown. Bioaccumulation is probably not an important environmental fate process. Although toluene is known to be degraded by microorganisms and can be detoxified and excreted by mammals, the available data do not allow estimation of the relative importance of biodegradation/biotransformation processes. Almost all toluene discharged to the environment by industry is in the form of atmospheric emissions.

Health Effects

There is no conclusive evidence that toluene is carcinogenic or mutagenic in animals or humans (USEPA 1980). The National Toxicological Program is currently conducting an inhalation carcinogenicity bioassay in rats and mice.

Oral administration of toluene at doses as low as 260 mg/kg produced a significant increase in embryonic lethality in mice (USEPA 1980). Decreased fetal weight was observed at doses as low as 434 mg/kg, and an increased incidence of cleft palate was seen at doses as low as 867 mg/kg. However, other researchers have reported that toluene is embryotoxic but not teratogenic in laboratory animals. There are no accounts of a teratogenic effect in humans after exposure to toluene.

Acute exposure to toluene at concentrations of 375-1,500 mg/m³ produces central nervous system depression and narcosis in humans (ACGIH 1980). However, even exposure to quantities sufficient to produce unconsciousness fail to produce residual organ damage. The rat oral LD₅₀ value and inhalation LC₅₀ value are 5,000 mg/kg and 15,000 mg/m³, respectively. Chronic inhalation exposure to toluene at relatively high concentrations produces cerebellar degeneration and an irreversible encephalopathy in mammals.

Toluene in sufficient amounts appears to have the potential to alter significantly the metabolism and resulting bioactivity of certain chemicals. For example, coadministration of toluene along with benzene or styrene has been shown to suppress the metabolism of benzene or styrene in rats.

Toxicity to Wildlife and Domestic Animals

Of five freshwater species tested with toluene, the cladoceran Daphnia magna was most resistant to any acute effects (USEPA 1980). The EC₅₀ and LC₅₀ values for all five species range from 12,700 to 313,000 µg/liter. No chronic tests are available for freshwater species. The two freshwater algal species tested are relatively insensitive to toluene with EC₅₀ values of 245,000 µg/liter or greater being reported. For saltwater species, EC₅₀ and LC₅₀ values range from 3,700 µg/liter for the bay shrimp to 1,050 mg/liter for the Pacific oyster. The chronic value in an embryo-larval test for the sheepshead minnow is reported to be between 3,200 and 7,700 µg/liter, and the acute-chronic ratio is between 55 and 97. In several saltwater algal species and kelp, effects occur at toluene concentrations from 8,000 to more than 433,000 µg/liter.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria. However, EPA did report the lowest concentrations of toluene known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: 17,500 µg/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 6,300 µg/liter
Chronic toxicity: 5,000 µg/liter

Human Health

Criterion: 14.3 mg/liter

NIOSH Recommended Standards: 375 mg/m³ TWA
560 mg/m³ STEL

OSHA Standards: 750 mg/m³ TWA
1,120 mg/m³ Ceiling Level

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).
1980. Documentation of the Threshold Limit Values. 4th ed.
Cincinnati, Ohio. 488 pages
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1973. Criteria for a Recommended Standard--Occupational
Exposure to Toluene. Washington, D.C. DHEW Publication
No. (NIOSH) HSM 73-11023
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983
- NATIONAL RESEARCH COUNCIL (NRC). 1980. The Alkyl Benzenes.
National Academy Press, Washington, D.C.
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials.
4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Toluene. Office of Water Regu-
lations and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-075
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Toluene. Final Draft. Environmental
Criteria and Assessment Office, Cincinnati, Ohio. Sep-
tember 1984. ECAO-CIN-HO33
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

POLYCHLORINATED BIPHENYLS

Summary

Polychlorinated biphenyls (PCBs) are very persistent in the natural environment and are readily bioaccumulated. In humans, exposure to PCBs has been associated with chloracne, impairment of liver function, a variety of neurobehavioral symptoms, menstrual disorders, minor birth abnormalities, and an increased incidence of cancer. Experimental animals exposed to PCBs experienced an increased incidence of cancer; reproductive problems; neurobehavioral degradation; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological function. PCBs are often contaminated, and these contaminants may be much more toxic than the PCBs themselves.

Background Information

Polychlorinated biphenyls (PCBs) are complex mixtures of chemicals composed of two connected benzene rings with 1 to 10 chlorine atoms attached. The chemical, physical, and biological properties of these materials depend to a large degree on the amount and location of the chlorine atoms on the two benzene rings of each specific PCB and on the particular mixture of individual chlorobiphenyls that comprise the mixture.

CAS Number: 1336-36-3

Chemical Formula: $C_6H_5Cl_xC_6H_5Cl_x$

IUPAC Name: Specific for each polychlorinated biphenyl

Important Synonyms and Trade Names: PCBs, chlorinated biphenyls, polychlorobiphenyls, Aroclor, Kanechlor, Clophen

Chemical and Physical Properties

Molecular Weight: 189-399*

Boiling Point: 267°C and up*

Melting Point: 54-310°C*

*Increases with increasing chlorination.

Polychlorinated biphenyls

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Specific Gravity: 1.3 to 1.5 at 20°C*

Solubility in Water: 0.003-0.6 mg/liter

Solubility in Organics: Soluble in most common organic solvents

Log Octanol/Water Partition Coefficient: 4-6*

Vapor Pressure: 10^{-3} - 10^{-5} mm Hg at 20°C**

Henry's Law Constant: 10^{-3} to 10^{-5} atm m³/mole

Transport and Fate

The transport and fate of polychlorinated biphenyls has been studied extensively, and although individual chemicals vary in the rates at which processes occur, some generalizations can be made about PCBs as a class. PCBs are relatively inert, and therefore persistent, compounds, with low vapor pressures, low water solubility, and high log octanol/water partition coefficients. Despite their low vapor pressures, they have a high activity coefficient in water, which causes a higher rate of volatilization than might normally be expected. Volatilization and persistence account for the ubiquitous nature of PCBs in the environment. Adsorption to the organic material in soil or sediments is probably the major fate of at least the more heavily chlorinated PCBs. Once bound, the PCBs may persist for years with slow desorption providing continuous, low-level exposure to the surrounding locality. Bioaccumulation of PCBs also occurs, with most of the compound stored in the adipose tissue of the body. PCBs are degraded primarily by two routes. Less heavily chlorinated PCBs (mainly the mono-, di-, and trichlorinated PCBs) can be biodegraded by some soil microorganisms. PCBs with five or more chlorines are not measurably biodegraded. These heavier PCBs can be photolyzed by ultraviolet light. This process is extremely slow, but it may be the most important degradation process for these very persistent compounds.

Assessing the toxicity of PCBs is complicated by the fact that several different mixtures have been produced and distributed commercially and by the presence of highly toxic contaminants in some commercial mixtures. Some of these contaminants can be formed by combustion of PCBs or even by high-temperature treatment in service, so that used materials may be more toxic than the commercial mixtures whose toxicity has been studied.

-
- *Increases with increasing chlorination.
 - **Decreases with increasing chlorination.

Health Effects

In humans exposed to PCBs (in the workplace or via accidental contamination of food), reported adverse effects include chloracne (a long-lasting, disfiguring skin disease), impairment of liver function, a variety of neurobehavioral and affective symptoms, menstrual disorders, minor birth abnormalities, and probably increased incidence of cancer. Animals experimentally exposed to PCBs have shown most of the same symptoms, as well as impaired reproduction; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological functions. PCBs are carcinogenic in rats and mice and, in appropriate circumstances, enhance the effects of other carcinogens. Reproductive and neurobiological effects of PCBs have been reported in rhesus monkeys at the lowest dose level tested, 11 µg/kg body weight/day over a period of several months.

Toxicity to Wildlife and Domestic Animals

Polychlorinated biphenyls are bioaccumulated and can be biomagnified. Therefore, their toxicity increases with length of exposure and position of the exposed species on the food chain. The toxicity of the various PCB mixtures is also dependent on their composition. Because of the complexity of PCB toxicity, only general effects will be discussed here.

The 96-hour LC_{50} values for rainbow trout, bluegills, and channel catfish were around 20 mg/liter. The same species exposed for 10 to 20 days had LC_{50} values of about 0.1 mg/liter. Invertebrate species were also adversely affected, with some species having 7-day LC_{50} values as low as 1 µg/liter. In general, juvenile organisms appeared more susceptible to the effects of PCBs than either eggs or adults.

Three primary ways in which PCBs can affect terrestrial wildlife are outright mortality, adversely affecting reproduction, and changing behavior. PCB doses greater than 200 ppm in the diet or 10 mg/kg body weight (bw) caused some mortality in sensitive bird species exposed for several days. Doses around 1,500 ppm (diet) or about 100 mg/kg (bw) caused extensive mortality in these sensitive species. They generally caused some mortality in all species, with the level being dependent on the length of exposure and the particular PCB mixture. Some mammalian species are especially susceptible to PCBs. For example, mink died when fed as little as 5 ppm in the diet (equivalent to less than 1 mg/kg bw/day). PCBs caused lower egg production; deformities; decreased hatchability, growth, and survival; and some eggshell thinning in reproductive studies on chickens fed doses of 20 ppm in the diet (1 mg/kg bw). Mink fed 1 ppm in the diet (0.2 mg/kg bw) had lower reproductive success, and there are indications that an increased incidence

of premature births in some marine mammals was linked to PCB exposure. Behavioral effects on wildlife include increased activity, decreased avoidance response, and decreased nesting, all of which could significantly influence survival in the wild.

No toxic effects on domestic animals other than chickens were reported in the sources reviewed, but susceptible species would probably be affected in a similar manner to laboratory animals and wildlife.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 2 µg/liter
Chronic toxicity: 0.014 µg/liter

Saltwater

Acute toxicity: 10 µg/liter
Chronic toxicity: 0.030 µg/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of PCBs in water are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	0.79 ng/liter
10^{-6}	0.079 ng/liter
10^{-7}	0.0079 ng/liter

CAG Unit Risk (USEPA): $4.34 \text{ (mg/kg/day)}^{-1}$

NIOSH Recommended Standard: 1.0 µg/m^3 TWA

ACGIH Threshold Limit Value: 0.5 mg/m^3 TWA

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).
1980. Documentation of the Threshold Limit Values. 4th
ed. Cincinnati, Ohio. 488 pages

- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1978.
IARC Monographs on the Evaluation of Carcinogenic Risk
of Chemicals to Humans. Vol. 18: Polychlorinated Biphenyls
and Polybrominated Biphenyls. World Health Organization,
Lyon, France. Pp. 43-103
- NATIONAL ACADEMY OF SCIENCES (NAS). 1977. Drinking Water
and Health. Safe Drinking Water Committee, Washington, D.C.
939 pages
- ROBERTS, J.R., RODGERS, D.W., BAILEY, J.R., and RORKE, M.A.
1978. Polychlorinated Biphenyls: Biological Criteria
for an Assessment of their Effects on Environmental Quality.
National Research Council of Canada, Ottawa, Canada.
NRCC No. 16077
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1976. National
Conference on Polychlorinated Biphenyls (November 19-21,
1975, Chicago, Illinois). Office of Toxic Substances,
Washington, D.C. March 1976. EPA 560/6-75-004
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Polychlorinated Biphenyls (PCBs).
Office of Water Regulations and Standards, Criteria and
Standards Division, Washington, D.C. October 1980. EPA
440/5-80-054
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Polychlorinated Biphenyls. Environmental
Criteria and Assessment Office, Cincinnati, Ohio. September
1984. ECAO-CIN-H004 (Final Draft)
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health
Assessment Document for Dichloromethane (Methylene Chloride).
Office of Health and Environmental Assessment. Washington,
D.C. February 1985. EPA 600/8-82/004F

BARIUM

Summary

In its pure form, barium is an extremely reactive metal that decomposes in water. In natural waters it forms insoluble carbonate or sulfate salts and is usually present at concentrations of less than 1 mg/liter. Insoluble forms of barium are not very toxic; but soluble barium salts are highly toxic after acute exposure, and they have a prolonged stimulant effect on muscles. A benign pneumoconiosis, baritosis, can result from inhaling barium dusts. The EPA Interim Primary Drinking Water Standard is 1 mg/liter.

CAS Number: 7440-39-3

Chemical Formula: Ba

IUPAC Name: Barium

Chemical and Physical Properties

Atomic Weight: 137.3

Boiling Point: 1,640°C

Melting Point: 725°C

Specific Gravity: 3.5

Solubility in Water: Decomposes; combines with sulfate present in natural waters to form BaSO_4 , which has a solubility of 1.6 mg/liter at 20°C

Solubility in Organics: Soluble in alcohol; insoluble in benzene

Transport and Fate

Barium is extremely reactive, decomposes in water, and readily forms insoluble carbonate and sulfate salts. Barium is generally present in solution in surface or groundwater only in trace amounts. Large amounts will not dissolve because natural waters usually contain sulfate, and the solubility of barium sulfate is generally low. Barium is not soluble at more than a few parts per million in water that contains sulfate at more than a few parts per million. However, barium sulfate may become considerably more soluble in the presence

of chloride and other anions. Monitoring programs show that it is rare to find barium in drinking water at concentrations greater than 1 mg/liter. Atmospheric transport of barium, in the form of particulates, can occur. Bioaccumulation is not an important process for barium.

Health Effects

There are no reports of carcinogenicity, mutagenicity, or teratogenicity associated with exposure to barium or its compounds. Effects on gametogenesis and on the reproductive organs are reported in male and female rats after inhalation of barium carbonate; intratesticular injection of barium chloride affects the male reproductive organs.

Insoluble forms of barium, particularly barium sulfate, are not toxic by ingestion or inhalation because only minimal amounts are absorbed. However, soluble barium compounds are highly toxic in humans after exposure by either route. The most important effect of acute barium poisoning is a strong, prolonged stimulant action on muscle. Smooth, cardiac, and skeletal muscles are all affected, and a transient increase in blood pressure due to vasoconstriction can occur. Effects on the hematopoietic system and cerebral cortex have also been reported in humans. Accidental ingestion of soluble barium salts has resulted in gastroenteritis, muscular paralysis, and ventricular fibrillation and extra systoles. Potassium deficiency can occur in cases of acute poisoning. Doses of barium carbonate and barium chloride of 57 mg/kg and 11.4 mg/kg, respectively, have been reported to be fatal in humans. Digitalis-like toxicity, muscle stimulation, and effects on the hematopoietic and central nervous systems have been confirmed in experimental animals. There are no adequate animal data available for determining the chronic effects of low level exposure to barium by ingestion.

Baritosis, a benign pneumoconiosis, is an occupational disease arising from the inhalation of barium sulfate dust, barium oxide dust, and barium carbonate. The radiologic changes produced in the lungs are reversible with cessation of exposure. Other reports of industrial exposure to barium compounds describe pulmonary nodulation with or without a decrease in lung function. Dusts of barium oxide are considered potential agents of dermal and nasal irritation. The biological half-life for barium is less than 24 hours.

Toxicity to Wildlife and Domestic Animals

Adequate data for characterization of toxicity to wildlife and domestic animals are not available.

Regulations and Standards

Interim Primary Drinking Water Standard: 1 mg/liter

OSHA Standard: 0.5 mg/m³ (soluble compounds, as Ba)

ACGIH Threshold Limit Value: 0.5 mg/m³ (soluble compounds, as Ba)

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

DOULL, J., KLAASSEN, C.D., and AMDUR, M.O., eds. 1980. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. 778 pages

NATIONAL ACADEMY OF SCIENCES (NAS). 1977. Drinking Water and Health. Safe Drinking Water Committee, Washington, D.C. 939 pages

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1984. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. July 1984

SAX, N.I. 1975. Dangerous Properties of Industrial Materials. 4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Barium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO21 (Final Draft)

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2332 pages

CADMIUM

Summary

Cadmium is a metal that can be present in a variety of chemical forms in wastes or in the environment. Some forms are insoluble in water, but cadmium is relatively mobile in the aquatic environment. Cadmium is carcinogenic in animals exposed by inhalation and may also be in humans. It is uncertain whether it is carcinogenic in animals or humans exposed via ingestion. Cadmium is a known animal teratogen and reproductive toxin. It has chronic effects on the kidney, and background levels of human exposure are thought to provide only a relatively small margin of safety for these effects.

Background Information

Cadmium is a soft, bluish white metal that is obtained as a by-product from the treatment of the ores of copper, lead, and iron. Cadmium has a valence of +2 and has properties similar to those of zinc. Cadmium forms both organic and inorganic compounds. Cadmium sulfate is the most common salt.

CAS Number: 7440-43-9

Chemical Formula: Cd

IUPAC Name: Cadmium

Chemical and Physical Properties

Atomic Weight: 112.41

Boiling Point: 765°C

Melting Point: 321°C

Specific Gravity: 8.642

Solubility in Water: Salts are water soluble; metal is insoluble

Solubility in Organics: Variable, based on compound

Vapor Pressure: 1 mm Hg at 394°C

Transport and Fate

Cadmium is relatively mobile in the aquatic environment compared to other heavy metals (USEPA 1979). It is removed from aqueous media by complexing with organic materials and subsequently being adsorbed to the sediment. It appears that cadmium moves slowly through soil, but only limited information on soil transport is available. Cadmium uptake by plants is not a significant mechanism for depletion of soil accumulations but may be significant for human exposure.

Health Effects

There is suggestive evidence linking cadmium with cancer of the prostate in humans (USEPA 1980). In animal studies, exposure to cadmium by inhalation caused lung tumors in rats, and exposure by injection produced injection-site sarcomas and/or Leydig-cell tumors (Takenaka 1983, USEPA 1981). An increased incidence of tumors has not been seen in animals exposed to cadmium orally, but four of the five available studies were inadequate by current standards (Clement 1983).

The evidence from a large number of studies on the mutagenicity of cadmium is equivocal, and it has been hypothesized that cadmium is not directly mutagenic but impedes repair (Clement 1983). Cadmium is a known animal teratogen and reproductive toxin. It has been shown to cause renal dysfunction in both humans and animals. Other toxic effects attributed to cadmium include immunosuppression (in animals), anemia (in humans), pulmonary disease (in humans), possible effects on the endocrine system, defects in sensory function, and bone damage. The oral LD₅₀ in the rat was 225 mg/kg (NIOSH 1983).

Toxicity to Wildlife and Domestic Animals

Laboratory experiments suggest that cadmium may have adverse effects on reproduction in fish at levels present in lightly to moderately polluted waters.

The acute LC₅₀ for freshwater fish and invertebrates generally ranged from 100 to 1,000 µg/liter; salmonids are much more sensitive than other organisms (USEPA 1980). Saltwater species were in general 10-fold more tolerant to the acute effects of cadmium. Chronic tests have been performed and show that cadmium has cumulative toxicity and acute-chronic ratios that range of from 66 to 431. Bioconcentration factors were generally less than 1,000 but were as high as 10,000 for some freshwater fish species.

No adverse effects on domestic or wild animals were reported in the studies reviewed.

Cadmium

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Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed 1984)

Freshwater

Acute toxicity: $e^{(1.30[\ln(\text{hardness})] - 3.92)} \mu\text{g/liter}$

Chronic toxicity: $e^{(0.87[\ln(\text{hardness})] - 4.38)} \mu\text{g/liter}$

Saltwater

Acute toxicity: 38 $\mu\text{g/liter}$

Chronic toxicity: 12 $\mu\text{g/liter}$

Human Health

Criterion: 10 $\mu\text{g/liter}$

CAG Unit Risk for inhalation exposure (USEPA): $6.1 (\text{mg/kg/day})^{-1}$

Interim Primary Drinking Water Standard (USEPA): 10 $\mu\text{g/liter}$

NIOSH Recommended Standards: 40 $\mu\text{g/m}^3$ TWA
200 $\mu\text{g/m}^3$ /15 min Ceiling Level

OSHA Standards: 200 $\mu\text{g/m}^3$ TWA
600 $\mu\text{g/m}^3$ Ceiling Level

ACGIH Threshold Limit Values: 50 $\mu\text{g/m}^3$ TWA

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).
1980. Documentation of the Threshold Limit Values. 4th
ed. Cincinnati, Ohio. 488 pages

CLEMENT ASSOCIATES, INC. 1983. Assessment of the Weight of
Evidence for Risk Assessment for Four Selected Toxic Air
Pollutants. Report Prepared for the Air Economic Branch,
OPRM, U.S. Environmental Protection Agency. May 1983.

FLEISCHER, M., SAROFIM, A.F., FASSETT, D.W., HAMMOND, P.,
SCHAKKETTE, H.T., NISBET, I.C.T., and EPSTEIN, S. 1974.
Environmental impact of cadmium: A review by the panel
on hazardous trace substances. Environ. Health. Perspect.
7:253-323

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983

TAKENAKA, S., OLDIGES, H., KONIG, H., HOCHRAINER, D., and
OBERDORSTER, G. 1983. Carcinogenicity of cadmium chloride
aerosols in W rats. JNCI 70:367-371

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Cadmium. Office of Water Regu-
lations and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-025

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1981. Health
Assessment Document for Cadmium. Environment Criteria
and Assessment Office. Research Triangle Park, North
Carolina. October 1981. EPA 600/8-81-023

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Cadmium. Environmental Criteria
and Assessment Office, Cincinnati, Ohio. September 1984.
ECAO-CIN-HO38 (Final Draft)

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health
Assessment Document for Chloroform. Office of Health
and Environmental Assessment, Washington, D.C. September
1985. EPA 600/8-84/004F

CHROMIUM

Summary

Chromium is a heavy metal that generally exists in either a trivalent or hexavalent oxidation state. Hexavalent chromium (Cr VI) is rather soluble and is quite mobile in groundwater and surface water. However, in the presence of reducing agents it is rapidly converted to trivalent chromium (Cr III), which is strongly adsorbed to soil components and consequently is much less mobile. A number of salts of hexavalent chromium are carcinogenic in rats. In addition, an increased incidence of lung cancer was seen in workers occupationally exposed to chromium VI. Hexavalent chromium also causes kidney damage in animals and humans. Trivalent chromium is less toxic than hexavalent chromium; its main effect is contact dermatitis in sensitive individuals.

CAS Number: 7440-47-3

Chemical Formula: Cr

IUPAC Name: Chromium

Chemical and Physical Properties (Metal)

Atomic Weight: 51.996

Boiling Point: 2672°C

Melting Point: 1857 \pm 20°C

Specific Gravity: 7.20 at 28°C

Solubility in Water: Insoluble; some compounds are soluble

Transport and Fate

Hexavalent Cr is quite soluble, existing in solution as a component of a complex anion. It is not sorbed to any significant degree by clays or hydrous metal oxides. The anionic form varies according to pH and may be a chromate, hydrochromate, or dichromate. Because all anionic forms are so soluble, they are quite mobile in the aquatic environment. Cr VI is efficiently

removed by activated carbon and thus may have some affinity for organic materials in natural water. Cr VI is a moderately strong oxidizing agent and reacts with reducing materials to form trivalent chromium. Most Cr III in the aquatic environment is hydrolyzed and precipitates as chromium hydroxide. Sorption to sediments and bioaccumulation will remove much of the remaining Cr III from solution. Cr III is adsorbed only weakly to inorganic materials. Cr III and Cr VI are readily interconvertible in nature depending on microenvironmental conditions such as pH, hardness, and the types of other compounds present. Soluble forms of chromium accumulate if ambient conditions favor Cr VI. Conditions favorable for conversion to Cr III lead to precipitation and adsorption of chromium in sediments.

In air, chromium is associated almost entirely with particulate matter. Sources of chromium in air include windblown soil and particulate emissions from industrial processes. Little information is available concerning the relative amounts of Cr III and Cr VI in various aerosols. Relatively small particles can form stable aerosols and can be transported many miles before settling out.

Cr III tends to be adsorbed strongly onto clay particles and organic particulate matter, but can be mobilized if it is complexed with organic molecules. Cr III present in minerals is mobilized to different extents depending on the weatherability and solubility of the mineral in which it is contained. Hexavalent compounds are not strongly adsorbed by soil components and Cr VI is mobile in groundwater. Cr VI is quickly reduced to Cr III in poorly drained soils having a high content of organic matter. Cr VI of natural origin is rarely found in soils.

Health Effects

The hexavalent form of chromium is of major toxicological importance in higher organisms. A variety of chromate (Cr VI) salts are carcinogenic in rats and an excess of lung cancer has been observed among workers in the chromate-producing industry. Cr VI compounds can cause DNA and chromosome damage in animals and humans, and Cr (VI) trioxide is teratogenic in the hamster. Inhalation of hexavalent chromium salts causes irritation and inflammation of the nasal mucosa, and ulceration and perforation of the nasal septum. Cr VI also produces kidney damage in animals and humans. The liver is also sensitive to the toxic effects of hexavalent Cr, but apparently less so than the kidneys or respiratory system. Cr III is less toxic than Cr VI; its main effect in humans is a form of contact dermatitis in sensitive individuals.

Toxicity to Wildlife and Domestic Animals

Chromium is an essential nutrient and is accumulated in a variety of aquatic and marine biota, especially benthic organisms, to levels much higher than in ambient water. Levels in biota, however, usually are lower than levels in the sediments. Passage of chromium through the food chain can be demonstrated. The food chain appears to be a more efficient pathway for chromium uptake than direct uptake from seawater.

Water hardness, temperature, dissolved oxygen, species, and age of the test organism all modify the toxic effects of chromium on aquatic life. Cr III appears to be more acutely toxic to fish than Cr VI; the reverse is true in long term chronic exposure studies.

None of the plants normally used as food or animal feed are chromium accumulators. Chromium absorbed by plants tends to remain primarily in the roots and is poorly translocated to the leaves. There is little tendency for chromium to accumulate along food chains in the trivalent inorganic form. Organic chromium compounds, about which little is known, can have significantly different bioaccumulation tendencies. Little information concerning the toxic effects of chromium on mammalian wildlife and domestic animal species is available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Cr VI:

Aquatic Life (Proposed Criteria)

Freshwater

Acute toxicity: 11 µg/liter
Chronic toxicity: 7.2 µg/liter

Saltwater

Acute toxicity: 1,200 µg/liter
Chronic toxicity: 54 µg/liter

Human Health

Criterion: 50 µg/liter

Cr III:

Aquatic Life (Proposed Criteria)

Freshwater

Acute toxicity: $e^{(0.819[\ln(\text{hardness})]+3.568)}$ $\mu\text{g/liter}$

Chronic toxicity: $e^{(0.819 [\ln(\text{hardness})])+0.537}$ $\mu\text{g/liter}$

Saltwater

The available data are not adequate for establishing criteria.

Human Health

Criterion: 170 mg/liter

CAG Unit Risk for inhalation exposure to CR VI (USEPA):
41 (mg/kg/day)⁻¹

National Interim Primary Drinking Water Standard: 50 $\mu\text{g/liter}$

NIOSH Recommended Standards for CR VI: 1 $\mu\text{g/m}^3$ carcinogenic
25 $\mu\text{g/m}^3$ noncarcinogenic TWA
50 $\mu\text{g/m}^3$ noncarcinogenic
(15-min sample)

OSHA Standards: OSHA air standards have been set for several chromium compounds. Most recognized or suspected carcinogenic chromium compounds have ceiling limits of 100 $\mu\text{g/m}^3$.

ACGIH Threshold Limit Values: Several chromium compounds have TWAs ranging from 0.05 to 0.5 mg/m^3 . Chromite ore processing (chromate), certain water insoluble Cr VI compounds, and chromates of lead and zinc are recognized or suspected human carcinogens and have 0.05 mg/m^3 TWAs.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. IARC Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 23: Some Metals and Metallic Compounds. World Health Organization, Lyon, France

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1975. Criteria for a Recommended Standard--Occupational
Exposure to Chromium (VI). Washington, D.C. DHEW Publi-
cation No. (NIOSH) 76-129

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983

NATIONAL RESEARCH COUNCIL OF CANADA. 1976. Effects of Chromium
in the Canadian Environment. Subcommittee on Heavy Metals
and Certain Other Compounds, Ottawa, Canada. Environmental
Secretariat Publication No. NRCC 15017

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Fate of 129 Priority Pollutants. Washington,
D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Chromium. Office of Water
Regulations and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-035

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Water
quality criteria: Request for comments. (Proposed Criteria)
Fed. Reg. 49:4551-4553

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Trivalent Chromium. Environmental
Criteria and Assessment Office, Cincinnati, Ohio. September
1984. ECAO-CIN-HO35 (Final Draft)

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Hexavalent Chromium. Environmental
Criteria and Assessment Office, Cincinnati, Ohio. September
1984. ECAO-CIN-HO19 (Final Draft)

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health
Assessment Document for Dichloromethane (Methylene Chloride).
Office of Health and Environmental Assessment. Washington,
D.C. February 1985. EPA 600/8-82/004F

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

COPPER

Summary

Copper is among the more mobile metals in the environment. It is toxic to humans at high levels; it causes irritation following acute exposure and anemia following chronic exposure. Sheep are very susceptible to copper toxicosis, as are many aquatic organisms.

Background Information

Copper exists in a valence state of +1 or +2. It is a lustrous, reddish metal. The physical properties of copper include ductility and conductivity of heat and electricity. Copper is found in nature as sulfide, oxide, or carbonate ore.

CAS Number: 7440-50-8

Chemical Formula: Cu

IUPAC Name: Copper

Chemical and Physical Properties

Atomic Weight: 63.546

Boiling Point: 2,567°C

Melting Point: 1,083°C

Specific Gravity: 8.92

Solubility in Water: Most copper salts are insoluble, with the exception of CuSO_4 , $\text{Cu}(\text{NO}_3)_2$, and CuCl_2 (the more common copper salts). The metal is insoluble in water.

Vapor Pressure: 1 mm Hg at 1,628°C

Transport and Fate

Copper has two oxidation states, +1 (cuprous) and +2 (cupric). Cuprous copper is unstable in aerated water over the pH range of most natural waters (6 to 8) and oxidizes to the cupric state. Several processes determine the fate of copper in the aquatic environment: formation of complexes, especially with humic substances; sorption to hydrous metal oxides, clays, and organic materials; and bioaccumulation. In waters polluted

with soluble organic material, complexation with organic ligands can occur, thus favoring the prolonged dispersion of copper in solution. The presence of organic acids also can lead to the mobilization of copper from the sediments to solution. Copper has a strong affinity for hydrous iron and manganese oxides, clays, carbonate minerals, and organic matter. Sorption to these materials, both suspended in the water column and in the sediment, results in relative enrichment of the solid phase and reduction in dissolved levels. Sorption processes are quite efficient in scavenging dissolved copper and in controlling its mobility in natural unpolluted streams. The amounts of the various copper compounds and complexes that actually exist in solution depend on the pH, temperature, alkalinity, and concentrations of other chemical species. The levels of copper able to remain in solution are directly dependent on water chemistry. Generally, ionic copper is more soluble in low pH waters and less soluble in high pH waters.

As an essential nutrient, copper is accumulated by plants and animals, although apparently it is not generally biomagnified. Because copper is strongly bioaccumulated and because biogenic ligands play an important role in complexing copper, biological activity is a major factor in determining the distribution and occurrence of copper in the ecosystem. For example, bioaccumulation patterns may exhibit seasonal variations related to biological activity.

Because many copper compounds and complexes are readily soluble, copper is among the more mobile heavy metals in soil and other surface environments. The major process that limits the environmental mobility of copper is adsorption to organic matter, clays, and other materials. Atmospheric transport of copper compounds can also occur.

Health Effects

Copper appears to increase the mutagenic activity of triose reductone and ascorbic acid in bacterial test systems. However, copper itself does not appear to have mutagenic, teratogenic, or carcinogenic effects in animals or humans. Dietary levels of trace elements such as molybdenum, sulfur, zinc, and iron can affect the level of copper that produces certain deficiency or toxicity symptoms. In general, more attention is given to the problems associated with copper deficiency than to problems of excess copper in the environment. However, high levels of copper can be toxic to humans.

Exposure to metallic copper dust can cause a short-term illness similar to metal fume fever that is characterized by chills, fever, aching muscles, dryness of mouth and throat, and headache. Exposure to copper fumes can produce upper

respiratory tract irritation, a metallic or sweet taste, nausea, metal fume fever, and sometimes discoloration of skin and hair. Individuals exposed to dusts and mists of copper salts may exhibit congestion of nasal mucous membranes, sometimes of the pharynx, and occasionally ulceration with perforation of the nasal septum.

If sufficient concentrations of copper salts reach the gastrointestinal tract, they act as irritants and can produce salivation, nausea, vomiting, gastritis, and diarrhea. Elimination of ingested ionic copper by vomiting and diarrhea generally protects the patient from more serious systemic toxic effects, which can include hemolysis, hepatic necrosis, gastrointestinal bleeding, oliguria, azotemia, hemoglobinuria, hematuria, proteinuria, hypotension, tachycardia, convulsions, and death. Chronic exposure may result in anemia.

Copper salts act as skin irritants producing an itching eczema. Conjunctivitis or even ulceration and turbidity of the cornea may result from direct contact of ionic copper with the eye.

Toxicity to Wildlife and Domestic Animals

Mean acute toxicity values for a large number of freshwater animals range from 7.2 µg/liter for Daphnia pulicaria to 10,200 µg/liter for the bluegill. Toxicity tends to decrease as hardness, alkalinity, and total organic carbon increase. Chronic values for a variety of freshwater species range from 3.9 µg/liter for brook trout to 60.4 µg/liter for northern pike. Hardness does not appear to affect chronic toxicity. The acute-chronic ratios for different species range from 3 to 156. The more sensitive species tend to have lower ratios than the less sensitive species. In addition, the ratio seems to increase with hardness. Acute toxicity values for saltwater organisms range from 17 µg/liter for a calanoid copepod to 600 µg/liter for the shore crab. A chronic value of 54 µg/liter and an acute-chronic ratio of 3.4 is reported for the mysid shrimp. Long-term exposure to 5 µg/liter is fatal to the bay scallop.

Bioconcentration factors in freshwater species range from zero for the bluegill to 2,000 for the alga Chlorella regularis. Among saltwater species, the highest bioaccumulation factors are those for the bivalve molluscs. Oysters can bioaccumulate copper up to 28,200 times without any significant mortality.

Sheep are very susceptible to copper toxicosis, and poisoning may be acute or chronic. Acute poisoning is caused by direct action of copper salts on the gastrointestinal tract, resulting in gastroenteritis, shock, and death. The toxic dose is about 200 mg/kg and is usually obtained through an

accidental overdosage of an antihelminthic. Ingestion of excess copper over a long period of time results in absorption and accumulation of copper by the liver. This type of chronic cumulative poisoning may suddenly develop into an acute hemolytic crisis. Copper intake of 1.5 g/day for 30 days is known to be fatal for many breeds of sheep. Excessive copper may be stored in the liver as a result of excess copper ingestion, as a consequence of impaired liver function, or in connection with a deficiency or excess of other trace elements. Sheep eliminate accumulated copper very slowly after cessation of exposure.

Swine develop copper poisoning at levels of 250 mg/kg in the diet unless zinc and iron levels are increased. Toxicosis develops with hypochromic microcytic anemia, jaundice, and marked increases in liver and serum copper levels as well as serum aspartate amino transferase. High copper levels may be found in swine because of the practice of feeding them high copper diets in order to increase daily weight gain. However, swine rapidly eliminate copper once it is removed from the diet. Cattle are much more resistant to copper in the diet than sheep or swine. Copper toxicity in ruminants can be counteracted by including molybdenum and sulfate in the diet.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed)

Freshwater

Acute toxicity: $e^{(0.905 [\ln(\text{hardness})] - 1.413)}$ $\mu\text{g/liter}$

Chronic toxicity: $e^{(0.905 [\ln(\text{hardness})] - 1.785)}$ $\mu\text{g/liter}$

Saltwater

Acute toxicity: 3.2 $\mu\text{g/liter}$

Chronic toxicity: 2.0 $\mu\text{g/liter}$

Human Health

Organoleptic criterion: 1 mg/liter

National Secondary Drinking Water Standards (USEPA): 1 mg/liter

OSHA Standards: 1.0 mg/m^3 TWA (dust and mist)
0.1 mg/m^3 TWA (fume)

ACGIH Threshold Limit Values: 1.0 mg/m³ TWA (dusts and mists)
0.2 mg/m³ TWA (fume)
2.0 mg/m³ STEL (dusts and mists)

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).
1980. Documentation of the Threshold Limit Values. 4th ed.
Cincinnati, Ohio. 488 pages
- BOSTWICK, J.L. 1982. Copper toxicosis in sheep. J. Am. Vet. Med.
Assoc. 180:386-387
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983
- UNDERWOOD, E.J. 1979. Trace metals in humans and animal health.
J. Hum. Nutr. 35:37-48
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Copper. Office of Water Regula-
tions and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-036
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Water
quality criteria, Request for comments. Fed. Reg. 49:4551-
4553
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Copper. Final Draft. Environmental
Criteria and Assessment Office, Cincinnati, Ohio. Sep-
tember 1984. ECAO-CIN-HO25
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

CYANIDE

Summary

Cyanide can be present in many forms in the environment. The transport, fate, and toxicity of the chemical is quite dependent on the specific form. Hydrogen cyanide and its simple salts are highly toxic following acute exposure by humans, experimental animals, and both aquatic and terrestrial wildlife.

Background Information

Cyanide (CN-) is usually defined as hydrogen cyanide (HCN) and its salts. The chemical/physical properties, transport and fate, and toxicity of cyanide are quite dependent on the form of cyanide present.

CAS Number: 151-50-8; 143-33-9

Chemical Formula: CN-

IUPAC Name: Cyanide

Chemical and Physical Properties

Molecular Weight: 27 (HCN)

Boiling Point: 26.7°C (HCN)

Melting Point: -14°C (HCN)

Specific Gravity: 0.699 at 22°C (HCN)

Solubility in Water: Soluble (HCN)

Solubility in Organics: Soluble in alcohol and ether

Vapor Pressure: 657.8 mm Hg at 21.9°C (HCN)

Transport and Fate

The transport and fate of cyanide in the environment is dependent on the chemical compound containing the cyanide. Most free cyanide will be HCN in aquatic environments and will probably evaporate, although biodegradation is another possible fate process. Metal cyanides are generally insoluble and for

that reason will accumulate in the sediment. Sorption occurs but is not considered an important transport or fate process. Cyanides move rather freely in soils but biodegradation would probably significantly decrease the amount present in the groundwater. Volatilization of HCN and nitriles may occur from soil surfaces.

Health Effects

Hydrogen cyanide and its simple salts, such as sodium cyanide, are highly toxic by all routes. Many reports are available regarding acute poisoning in humans. Hydrogen cyanide vapor is irritating at very low concentrations, is considered dangerous at 20 ppm (20 mg/m³), and is fatal at concentrations of 100 ppm (100 mg/m³) for one hour. NIOSH notes reports of chronic poisoning resulting in fatigue, weariness and other subjective symptoms in workers, but these findings have been disputed by other investigators. Chronic exposure to low levels of cyanide salts has been reported to cause enlargement of the thyroid gland in humans, apparently due to inefficient elimination of the cyanide metabolite thiocyanate. NIOSH (1976) concluded that there was no evidence of carcinogenicity, mutagenicity, or teratogenicity for cyanides. Cyanide has been shown to produce chromosome breaks in a plant, Vicia faba. Because of its mechanism of action, inhibition of the electron transport system in oxidative phosphorylation, cyanide is acutely toxic to almost all forms of life. A reduction in the TLV for HCN from 10 mg/m³ to a ceiling value of 3 mg/m³ has been recommended by several investigators, to prevent the various nonspecific effects noted by several investigators (ACGIH 1980).

Toxicity to Wildlife and Domestic Animals

Cyanide is acutely toxic to both freshwater and saltwater organisms, causing death at levels of about 50 µg/liter in sensitive species and being fatal to many species at levels above 200 µg/liter. Final acute values were determined to be 44.7 µg/liter for freshwater species and 2.03 µg/liter for saltwater species. Effects such as reduced survival and reduced reproduction were seen in fish chronically exposed to free cyanide concentrations of from 10 to 50 µg/liter. The final acute chronic ratios were determined to be 10.7 and 3.5 for freshwater and saltwater organisms, respectively. The final chronic values were determined by dividing the acute values by the acute-chronic ratio, and were determined to be 4.2 for freshwater species and 0.57 for saltwater organisms. An accidental spill of cyanide caused the death of 4,800 fish in Oak Ridge, Tennessee. The long-term effects of this spill were not reported. Livestock death and environmental damage were caused by high levels of cyanide leaching from a drum disposal site in Illinois.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed)

Freshwater

Acute toxicity: 22 µg/liter
Chronic toxicity: 4.2 µg/liter

Saltwater

Acute toxicity: 1.0 µg/liter
Chronic toxicity: 0.57 µg/liter

Human Health

Criterion: 200 µg/liter

Primary Drinking Water Standard (USEPA): 200 µg/liter

ACGIH Threshold Limit Value: 5 mg/m³ TWA

REFERENCES

- AMERICAN COUNCIL OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).
1980. Documentation of Threshold Limit Values. 4th ed.
Cincinnati, Ohio. 488 pages
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1976. Criteria for a Recommended Standard--Occupational
Exposure to Hydrogen Cyanide and Cyanide Salts (NaCN,
KCN, and Ca(CN)₂). Washington, D.C. DHEW Publication
No. (NIOSH) 77-108
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants
Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Cyanides. Office of Water
Regulations and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-037.

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1983. Revised Section B of Ambient Water Criteria for Cyanide--Aquatic Toxicology. Draft Report. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. August 1983

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Cyanide. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H011 (Final Draft)

VERSCHUEREN, K. 1977. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Co., New York. 659 pages

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

LEAD

Summary

Lead is a heavy metal that exists in one of three oxidation states, 0, +2, and +4. There is suggestive evidence that some lead salts are carcinogenic, inducing kidney tumors in mice and rats. Lead is also a reproductive hazard, and it can adversely affect the brain and central nervous system by causing encephalopathy and peripheral neuropathy. Chronic exposure to low levels of lead can cause subtle learning disabilities in children. Exposure to lead can also cause kidney damage and anemia, and it may have adverse effects on the immune system.

CAS Number: 7439-92-1

Chemical Formula: Pb

IUPAC Name: Lead

Chemical and Physical Properties

Atomic Weight: 207.19

Boiling Point: 1,740°C

Melting Point: 327.502°C

Specific Gravity: 11.35 at 20°C

Solubility in Water: Insoluble; some organic compounds are soluble

Solubility in Organics: Soluble in HNO_3 and hot, concentrated H_2SO_4

Transport and Fate

Some industrially produced lead compounds are readily soluble in water (USEPA 1979). However, metallic lead and the common lead minerals are insoluble in water. Natural compounds of lead are not usually mobile in normal surface or groundwater because the lead leached from ores is adsorbed by ferric hydroxide or combines with carbonate or sulfate ions to form insoluble compounds.

Movement of lead and its inorganic and organolead compounds as particulates in the atmosphere is a major environmental transport process. Lead carried in the atmosphere can be removed by either wet or dry deposition. Although little evidence is available concerning the photolysis of lead compounds in natural waters, photolysis in the atmosphere occurs readily. These atmospheric processes are important in determining the form of lead entering aquatic and terrestrial systems.

The transport of lead in the aquatic environment is influenced by the speciation of the ion. Lead exists mainly as the divalent cation in most unpolluted waters and becomes adsorbed into particulate phases. However, in polluted waters organic complexation is most important. Volatilization of lead compounds probably is not important in most aquatic environments.

Sorption processes appear to exert a dominant effect on the distribution of lead in the environment. Adsorption to inorganic solids, organic materials, and hydrous iron and manganese oxides usually controls the mobility of lead and results in a strong partitioning of lead to the bed sediments in aquatic systems. The sorption mechanism most important in a particular system varies with geological setting, pH, Eh, availability of ligands, dissolved and particulate ion concentrations, salinity, and chemical composition. The equilibrium solubility of lead with carbonate, sulfate, and sulfide is low. Over most of the normal pH range, lead carbonate, and lead sulfate control solubility of lead in aerobic conditions, and lead sulfide and the metal control solubility in anaerobic conditions. Lead is strongly complexed to organic materials present in aquatic systems and soil. Lead in soil is not easily taken up by plants, and therefore its availability to terrestrial organisms is somewhat limited.

Bioaccumulation of lead has been demonstrated for a variety of organisms, and bioconcentration factors are within the range of 100-1,000. Microcosm studies indicate that lead is not biomagnified through the food chain. Biomethylation of lead by microorganisms can remobilize lead to the environment. The ultimate sink of lead is probably the deep oceans.

Health Effects

There is evidence that several lead salts are carcinogenic in mice or rats, causing tumors of the kidneys after either oral or parenteral administration. Data concerning the carcinogenicity of lead in humans are inconclusive. The available data are not sufficient to evaluate the carcinogenicity of organic lead compounds or metallic lead. There is equivocal evidence that exposure to lead causes genotoxicity in humans and animals. The available evidence indicates that lead presents

a hazard to reproduction and exerts a toxic effect on conception, pregnancy, and the fetus in humans and experimental animals (USEPA 1977, 1980).

Many lead compounds are sufficiently soluble in body fluids to be toxic (USEPA 1977, 1980). Exposure of humans or experimental animals to lead can result in toxic effects in the brain and central nervous system, the peripheral nervous system, the kidneys, and the hematopoietic system. Chronic exposure to inorganic lead by ingestion or inhalation can cause lead encephalopathy, and severe cases can result in permanent brain damage. Lead poisoning may cause peripheral neuropathy in adults and children, and permanent learning disabilities that are clinically undetectable in children may be caused by exposure to relatively low levels. Short-term exposure to lead can cause reversible kidney damage, but prolonged exposure at high concentrations may result in progressive kidney damage and possibly kidney failure. Anemia, due to inhibition of hemoglobin synthesis and a reduction in the life span of circulating red blood cells, is an early manifestation of lead poisoning. Several studies with experimental animals suggest that lead may interfere with various aspects of the immune response.

Toxicity to Wildlife and Domestic Animals

Freshwater vertebrates and invertebrates are more sensitive to lead in soft water than in hard water (USEPA 1980, 1983). At a hardness of about 50 mg/liter CaCO_3 , the median effect concentrations for nine families range from 140 $\mu\text{g/liter}$ to 236,600 $\mu\text{g/liter}$. Chronic values for Daphnia magna and the rainbow trout are 12.26 and 83.08 $\mu\text{g/liter}$, respectively, at a hardness of about 50 mg/liter. Acute-chronic ratios calculated for three freshwater species ranged from 18 to 62. Bioconcentration factors, ranging from 42 for young brook trout to 1,700 for a snail, were reported. Freshwater algae show an inhibition of growth at concentrations above 500 $\mu\text{g/liter}$.

Acute values for twelve saltwater species range from 476 $\mu\text{g/liter}$ for the common mussel to 27,000 $\mu\text{g/liter}$ for the soft-shell clam. Chronic exposure to lead causes adverse effects in mysid shrimp at 37 $\mu\text{g/liter}$, but not at 17 $\mu\text{g/liter}$. The acute-chronic ratio for this species is 118. Reported bioconcentration factors range from 17.5 for the Quahog clam to 2,570 for the blue mussel. Saltwater algae are adversely affected at approximate lead concentrations as low as 15.8 $\mu\text{g/liter}$.

Although lead is known to occur in the tissue of many free-living wild animals, including birds, mammals, fishes, and invertebrates, reports of poisoning usually involve waterfowl. There is evidence that lead, at concentrations occasionally found near roadsides and smelters, can eliminate or reduce

populations of bacteria and fungi on leaf surfaces and in soil. Many of these microorganisms play key roles in the decomposer food chain.

Cases of lead poisoning have been reported for a variety of domestic animals, including cattle, horses, dogs, and cats. Several types of anthropogenic sources are cited as the source of lead in these reports. Because of their curiosity and their indiscriminate eating habits, cattle experience the greatest incidence of lead toxicity among domestic animals.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed Criteria)

The concentrations below are for active lead, which is defined as the lead that passes through a 0.45- μ m membrane filter after the sample is acidified to pH 4 with nitric acid.

Freshwater

Acute toxicity: $e^{(1.34 [\ln(\text{hardness})] - 2.014)}$ μ g/liter

Chronic toxicity: $e^{(1.34 [\ln(\text{hardness})] - 5.245)}$ μ g/liter

Saltwater

Acute toxicity: 220 μ g/liter

Chronic toxicity: 8.6 μ g/liter

Human Health

Criterion: 50 μ g/liter

Primary Drinking Water Standard: 50 μ g/liter

NIOSH Recommended Standard: 0.10 mg/m³ TWA (inorganic lead)

OSHA Standard: 50 μ g/m³ TWA

ACGIH Threshold Limit Values:

0.15 mg/m³ TWA (inorganic dusts and fumes)

0.45 mg/m³ STEL (inorganic dusts and fumes)

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- DOULL, J., KLAASSEN, L.D., and AMDUR, M.O., eds. 1980. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. 778 pages
- INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol. 23: Some Metals and Metallic Compounds. World Health Organization, Lyon, France. Pp. 325-415
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- NRIAGU, J.O., ed. 1978. The Biogeochemistry of Lead in the Environment: Part B. Biological Effects. Elsevier/North-Holland Biomedical Press, New York. 397 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1977. Air Quality Criteria for Lead. Office of Research and Development, Washington, D.C. December 1977. EPA 600/8-77-017
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Lead. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-057
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1983. Draft Revised Section B of Ambient Water Quality Criteria for Lead. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. August 1983
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Lead. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO55
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages
- WORLD HEALTH ORGANIZATION. 1977. Environmental Health Criteria: 3. Lead. World Health Organization, Geneva. 160 pages

NICKEL

Summary

In a number of epidemiological studies, occupational exposure to nickel compounds has been associated with excess cancer of the lung and nasal cavity. In addition, inhalation exposure to nickel subsulfide and nickel carbonyl has been shown to cause cancer in rats, while studies of other nickel compounds administered to animals by other routes have reported carcinogenic effects as well. Several nickel compounds are mutagenic and can cause cell transformation. In humans, nickel and nickel compounds can cause a sensitization dermatitis. The chronic toxicity of nickel to aquatic organisms is high.

Background Information

The commonly occurring valences of nickel are 0, +1, +2, and +3, with +4 rarely encountered. Although elemental nickel is seldom found in nature and is not soluble in water, many nickel compounds are highly soluble in water. Nickel is almost always found in the divalent oxidation state in aquatic systems.

CAS Number: 7440-02-0

Chemical Formula: Ni

IUPAC Name: Nickel

Chemical and Physical Properties

Atomic Weight: 58.71

Boiling Point: 2,732°C

Melting Point: 1,453°C

Specific Gravity: 8.902 at 25°C

Solubility in Water: Insoluble; some salts are soluble

Solubility in Organics: Depends on the properties of the specific nickel salt

Vapor Pressure: 1 mm Hg at 1,810°C

Transport and Fate

Nickel is a highly mobile metal in aquatic systems because many nickel compounds are highly soluble in water. However, the insoluble sulfide is formed under reducing conditions and in the presence of sulfur. Above pH 9, precipitation of the hydroxide or carbonate exhibits some control on nickel mobility. In aerobic environments below pH 9, soluble compounds are formed with hydroxide, carbonate, sulfate, and organic ligands.

In natural, unpolluted waters, sorption and coprecipitation processes involving hydrous iron and manganese oxides are probably at least moderately effective in limiting the mobility of nickel. In more organic-rich, polluted waters, it appears that little sorption of nickel is likely. The lack of other controls on nickel mobility probably makes incorporation into bed sediments an important fate of nickel in surface waters. However, much of the nickel entering the aquatic environment will be transported to the oceans.

In general, nickel is not accumulated in significant amounts by aquatic organisms. Bioconcentration factors are usually on the order of 100 to 1,000. Uptake of nickel from the soil by plants can also occur. Photolysis, volatilization, and biotransformation are not important environmental fate processes for nickel. However, atmospheric transport of nickel and nickel compounds on particulate matter can occur.

Health Effects

There is extensive epidemiological evidence indicating excess cancer of the lung and nasal cavity for workers at nickel refineries and smelters, and weaker evidence for excess risk in workers at nickel electroplating and polishing operations. Respiratory tract cancers have occurred in excess at industrial facilities that are metallurgically diverse in their operations. The nickel compounds that have been implicated as having carcinogenic potential are insoluble dusts of nickel subsulfide and nickel oxides, the vapor of nickel carbonyl, and soluble aerosols of nickel sulfate, nitrate, or chloride. Inhalation studies with experimental animals suggest that nickel subsulfide and nickel carbonyl are carcinogenic in rats. Evidence for the carcinogenicity of nickel metal and other compounds is relatively weak or inconclusive. Studies with experimental animals indicate that nickel compounds can also produce various types of malignant tumors in experimental animals after administration by other routes, including subcutaneous, intramuscular, implantation, intravenous, intrarenal, and intrapleural. Carcinogenic potential is not strongly dependent on route or site of administration but appears to be inversely related to the solubility of the compounds in aqueous media. Insoluble compounds, such

as nickel dust, nickel sulfide, nickel carbonate, nickel oxide, nickel carbonyl, and nickelocene are carcinogenic, whereas soluble nickel salts such as nickel chloride, nickel sulfate, and nickel ammonium sulfate, are not.

Mammalian cell transformation data indicate that several nickel compounds are mutagenic and can cause chromosomal alterations. The available information is inadequate for assessing teratogenic and reproductive effects of nickel in humans and experimental animals.

Dermatitis and other dermatological effects are the most frequent effects of exposure to nickel and nickel-containing compounds. The dermatitis is a sensitization reaction. Most information regarding acute toxicity of nickel involves inhalation exposure to nickel carbonyl. Clinical manifestations of acute poisoning include both immediate and delayed symptoms. Acute chemical pneumonitis is produced, and death may occur at exposures of 30 ppm (107 mg/m³) for 30 minutes. Rhinitis, nasal sinusitis, and nasal mucosal injury are among the effects reported among workers chronically exposed to various nickel compounds. Studies with experimental animals suggest that nickel and nickel compounds have relatively low acute and chronic oral toxicity.

Toxicity to Wildlife and Domestic Animals

In freshwater, toxicity depends on hardness; nickel tends to be more toxic in softer water. Acute values for exposure to a variety of nickel salts, expressed as nickel, range from 510 µg/liter for Daphnia magna to 46,200 µg/liter for banded killifish at comparable hardness levels. Chronic values range from 14.8 µg/liter for Daphnia magna in soft water to 530 µg/liter for the fathead minnow in hard water. Acute-chronic ratios for Daphnia magna range from 14 in hard water to 83 in soft water, and are approximately 50 in both hard and soft water for the fathead minnow. Residue data for the fathead minnow indicate a bioconcentration factor of 61. Freshwater algae experience reduced growth at nickel concentrations as low as 100 µg/liter.

Acute values for saltwater species range from 152 µg/liter for mysid shrimp to 350,000 µg/liter for the mummichog. A chronic value of 92.7 µg/liter is reported for the mysid shrimp, which gives an acute-chronic ratio of 5.5 for the species. Reduced growth is seen in saltwater algae at concentrations as low as 1,000 µg/liter. Bioconcentration factors ranging from 299 to 416 have been reported for the oyster and mussel.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: $e^{(0.76 [\ln(\text{hardness})] + 4.02)} \mu\text{g/liter}$

Chronic toxicity: $e^{(0.76 [\ln(\text{hardness})] + 1.06)} \mu\text{g/liter}$

Saltwater

Acute toxicity: 140 $\mu\text{g/liter}$

Chronic toxicity: 7.1 $\mu\text{g/liter}$

Human Health

Criterion: 13.4 $\mu\text{g/liter}$

CAG Unit Risk (USEPA): $1.15 (\text{mg/kg/day})^{-1}$

NIOSH Recommended Standard: 15 $\mu\text{g/m}^3$ TWA (inorganic nickel)

OSHA Standard: 1 mg/m^3 (metal and soluble compounds, as nickel)

ACGIH Threshold Limit Values:

0.1 mg/m^3 TWA (soluble compounds, as nickel)

0.3 mg/m^3 STEL (soluble compounds, as nickel)

0.35 mg/m^3 TWA (nickel carbonyl, as nickel)

1 mg/m^3 TWA (nickel sulfide roasting, fume and dust, as nickel; human carcinogen)

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages

NATIONAL ACADEMY OF SCIENCES (NAS). 1975. Medical and Environmental Effects of Environmental Pollutants: Nickel. Committee on Medical and Biological Effects of Environmental Pollutants, Division of Medical Sciences, National Research Council, Washington, D.C. 277 pages

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Inorganic Nickel. Washington, D.C. May 1977. DHEW Publication No. (NIOSH) 77-164

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances.
Data Base. Washington, D.C. October 1983

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Nickel. Office of Water Regu-
lations and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-060

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Nickel. Final Draft. Environmental
Criteria and Assessment Office, Cincinnati, Ohio. September
1984. ECAO-CIN-H018

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health
Assessment Document for Dichloromethane (Methylene Chloride).
Office of Health and Environmental Assessment. Washington,
D.C. February 1985. EPA 600/8-82/004F

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

Summary

Both organic and inorganic forms of mercury are reported to be teratogenic and embryotoxic in experimental animals. In humans, prenatal exposure to methylmercury has been associated with brain damage. Other major target organs for organic mercury compounds in humans are the central and peripheral nervous system and the kidney. In animals, toxic effects also occur in the liver, heart, gonads, pancreas, and gastrointestinal tract. Inorganic mercury is generally less acutely toxic than organic mercury compounds, but it does affect the central nervous system adversely.

Background Information

Several forms of mercury, including insoluble elemental mercury, inorganic species, and organic species, can exist in the environment. In general, the mercurous (+1) salts are much less soluble than the more commonly found mercuric (+2) salts. Mercury also forms many stable organic complexes that are generally much more soluble in organic liquids than in water. The nature and solubility of the chemical species that occur in an environmental system depend on the redox potential and the pH of the environment.

CAS Number: 7439-97-6

Chemical Formula: Hg

IUPAC Name: Mercury

Chemical and Physical Properties (Metal)

Atomic Weight: 200.59

Boiling Point: 356.58°C

Melting Point: -38.87°C

Specific Gravity: 13.5939 at 20°C

Solubility in Water: 81.3 µg/liter at 30°C; some salts and organic compounds are soluble

Solubility in Organics: Depends on chemical species

Vapor Pressure: 0.0012 mm Hg at 20°C

Transport and Fate

Mercury and certain of its compounds, including several inorganic species and dimethyl mercury, can volatilize to the atmosphere from aquatic and terrestrial sources. Volatilization is reduced by conversion of metallic mercury to complexed species and by deposition of HgS in reducing sediments, but even so atmospheric transport is the major environmental distribution pathway for mercury. Precipitation is the primary mechanism for removal of mercury from the atmosphere. Photolysis is important in the breakdown of airborne mercurials and may be important in some aquatic systems. Adsorption onto suspended and bed sediments is probably the most important process determining the fate of mercury in the aquatic environment. Sorption is strongest into organic materials. Mercury in soils is generally complexed to organic compounds.

Virtually any mercury compound can be remobilized in aquatic systems by microbial conversion to methyl and dimethyl forms. Conditions reported to enhance biomethylation include large amounts of available mercury, large numbers of bacteria, the absence of strong complexing agents, near neutral pH, high temperatures, and moderately aerobic environments. Mercury is strongly bioaccumulated by numerous mechanisms. Methylmercury is the most readily accumulated and retained form of mercury in aquatic biota, and once it enters a biological system it is very difficult to eliminate.

Health Effects

When administered by intraperitoneal injection, metallic mercury produces implantation site sarcomas in rats. No other studies were found connecting mercury exposure with carcinogenic effects in animals or humans. Several mercury compounds exhibit a variety of genotoxic effects in eukaryotes. In general, organic mercury compounds are more toxic than inorganic compounds. Although brain damage due to prenatal exposure to methylmercury has occurred in human populations, no conclusive evidence is available to suggest that mercury causes anatomical defects in humans. Embryotoxicity and teratogenicity of methylmercury has been reported for a variety of experimental animals. Mercuric chloride is reported to be teratogenic in experimental animals. No conclusive results concerning the teratogenic effects of mercury vapor are available.

In humans, all mercury compounds pass through the blood brain barrier and the placenta very rapidly, in contrast to inorganic mercury compounds. Major target organs are the central and peripheral nervous systems, and the kidney. Methylmercury is particularly hazardous because of the difficulty of eliminating it from the body. In experimental animals, organic mercury compounds can produce toxic effects in the gastrointestinal tract, pancreas, liver, heart, and gonads, with involvement of the endocrine, immunocompetent, and central nervous systems.

Elemental mercury is not highly toxic as an acute poison. However, inhalation of high concentrations of mercury vapor can cause pneumonitis, bronchitis, chest pains, dyspnea, coughing, stomatitis, gingivitis, salivation, and diarrhea. Soluble mercuric salts are highly poisonous on ingestion, with oral LD₅₀ values of 20 to 60 mg/kg reported. Mercurous compounds are less toxic when administered orally. Acute exposure to mercury compounds at high concentrations causes a variety of gastrointestinal symptoms and severe anuria with uremia. Signs and symptoms associated with chronic exposure involve the central nervous system and include behavioral and neurological disturbances.

Toxicity to Wildlife and Domestic Animals

The toxicity of mercury compounds has been tested in a wide variety of aquatic organisms. Although methylmercury appears to be more toxic than inorganic mercuric salts, few acute or chronic toxicity tests have been conducted with it. Among freshwater species, the 96-hour LC₅₀ values for inorganic mercuric salts range from 0.02 µg/liter for crayfish to 2,000 µg/liter for caddisfly larvae. Acute values for methylmercuric compounds and other mercury compounds are only available for fishes. In rainbow trout, methylmercuric chloride is about ten times more toxic to rainbow trout than mercuric chloride, which is acutely toxic at about 300 µg/liter at 10°C. Methylmercury is the most chronically toxic of the tested compounds, with chronic values for Daphnia magna and brook trout of 1.00 and 0.52 µg/liter, respectively. The acute-chronic ratio for Daphnia magna is 3.2.

Mean acute values for saltwater species range from 3.5 to 1,680 µg/liter. In general, molluscs and crustaceans are more sensitive than fish to the acute toxic effects of mercury. A life-cycle experiment with the mysid shrimp showed that inorganic mercury at a concentration of 1.6 µg/liter significantly influences time of appearance of first brood, time of first spawn, and productivity. The acute-chronic ratio for the mysid shrimp is 2.9.

Chronic dietary exposure of chickens to mercuric chloride at growth inhibitory levels causes immune suppression, with a differential reduction effect on specific immunoglobulins.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life (Proposed Criteria)

Freshwater

Acute toxicity: 1.1 µg/liter
Chronic toxicity: 0.20 µg/liter

Saltwater

Acute toxicity: 1.9 µg/liter
Chronic toxicity: 0.10 µg/liter

Human Health

Criterion: 144 ng/liter

Primary Drinking Water Standard: 0.002 mg/liter

NIOSH Recommended Standard: 0.05 mg/m³ TWA (inorganic mercury)

OSHA Standard: 0.1 mg/m³ Ceiling Level

ACGIH Threshold Limit Values:

0.01 mg/m³ TWA (alkyl compounds)
0.03 mg/m³ STEL (alkyl compounds)
0.05 mg/m³ TWA (vapor)
0.1 mg/m³ TWA (aryl and inorganic compounds)

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH).
1980. Documentation of the Threshold Limit Values. 4th
ed. Cincinnati, Ohio. 488 pages

BRIDGER, M.A., and THAXTON, J.P. 1983. Humoral immunity in
the chicken as affected by mercury. Arch. Environ. Contam.
Toxicol. 12:45-49

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH).
1983. Registry of Toxic Effects of Chemical Substances
Data Base. Washington, D.C. October 1983

SHEPARD, T.H. 1980. Catalog of Teratogenic Agents. 3rd ed.
Johns Hopkins University Press, Baltimore. 410 pages

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-
Related Environmental Fate of 129 Priority Pollutants.
Washington, D.C. December 1979. EPA 440/4-79-029

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient
Water Quality Criteria for Mercury. Office of Water Regu-
lations and Standards, Criteria and Standards Division,
Washington, D.C. October 1980. EPA 440/5-80-058

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Water
quality criteria; Request for comments. Fed. Reg. 49:
4551-4553

U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health
Effects Assessment for Mercury. Environmental Criteria
and Assessment Office, Cincinnati, Ohio. September 1984.
ECAO-CIN-HO42 (Final Draft)

WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics.
62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

WORLD HEALTH ORGANIZATION (WHO). 1976. Environmental Health
Criteria: 1. Mercury. World Health Organization, Geneva.
131 pages

SILVER

Summary

Exposure to high levels of silver can cause argyria (an impregnation of the tissues) and lesions of the liver, kidney, bone marrow, and lungs in humans. Liver and kidney damage, central nervous system effects, and pulmonary edema and congestion have been reported in experimental animals exposed to various silver compounds.

CAS Number: 7440-22-4

Chemical Formula: Ag

IUPAC Name: Silver

Chemical and Physical Properties

Atomic Weight: 107.868

Boiling Point: 2212°C

Melting Point: 961.93°C

Specific Gravity: 10.5 at 20°C

Solubility in Water: Insoluble (some compounds are soluble)

Solubility in Organics: Soluble in alkali cyanide solutions

Transport and Fate

Silver can exist in several chemical forms in aqueous systems. Metallic silver, which has very low solubility, is stable over much of the Eh-pH range for water. Concentrations of hydrated silver cations, usually present as the univalent species, may be controlled by reaction with chloride, bromide, and iodide ions to give insoluble silver halides. Precipitation of AgCl may exert a major control on solubility of silver where chloride concentrations are relatively high. Under the reducing conditions often found in bed sediments, formation of insoluble silver sulfides and metallic silver may also control levels of soluble silver species. Silver is strongly sorbed by manganese dioxide, ferric hydroxide, and clay minerals. Sorption is probably the dominant process leading to removal of dissolved

silver from the water column. In general, concentrations of silver are higher in the bed sediments than in overlying waters. For example, these concentrations were reported to differ by a factor of 1,000 in an alpine lake.

Bioaccumulation of silver by aquatic plants, invertebrates, and vertebrates occurs readily and appears to depend primarily on sorption/desorption from sediments. However, the amount of silver partitioned to the biota appears to be minor in comparison with the amount partitioned to the sediments. Little food-chain magnification seems to occur. Photolysis, volatilization, atmospheric transport, and biotransformation do not appear to be important fate or transport processes for silver.

Health Effects

Only equivocal evidence exists to suggest that silver has carcinogenic activity in experimental animals. Silver implants and injected colloidal suspensions are reported to produce tumors or hyperplasia at the site of application in several studies. However, it is suggested that the effects are due to the physical form of the metal or to its action as an exogenous irritant. There are no studies to suggest that silver is carcinogenic in humans. Silver does not appear to have significant mutagenic or teratogenic activity in humans or experimental animals.

Silver can be absorbed in humans by inhalation or ingestion. The most common and most noticeable effects of excessive absorption are a local or generalized impregnation of the tissues referred to as argyria. In cases of argyria, accumulation of silver can result in a blue-gray pigmentation of the skin, hair, internal organs, and conjunctiva of the eye. Large oral doses of silver compounds may produce serious effects in humans. For example, silver nitrate can cause violent abdominal pain, vomiting, and convulsions, and ingestion of 10 grams is reported to usually be fatal. Lesions of the liver, kidney, bone marrow, and lungs have also been attributed to industrial or medicinal exposure.

Intravenous administration of silver nitrate is reported to produce pulmonary edema and congestion in experimental animals. Liver and kidney damage, central nervous system effects, and death have also been reported in experimental animals exposed to various silver compounds. The intraperitoneal LD₅₀ (30 days) for Ag⁺ as the nitrate in male Swiss albino mice is 13.9 mg/kg. Rats exposed to silver in their drinking water for 11 months showed no toxic effects at concentrations less than 0.4 mg/liter. Hemorrhaging occurred in the kidneys at 0.4 mg/liter. Conditioned reflex activity and immunological resistance were lowered, and brain nucleic acid content was increased at 0.5 mg/liter.

Numerous physiological changes, including growth depression, and pathomorphological changes in the liver, kidney, stomach, and small intestine were evident in rats exposed to 20 mg/liter for 5 months.

Toxicity to Wildlife and Domestic Animals

Acute toxicity values for freshwater invertebrates range from 0.25 µg/liter for Daphnia magna to 4,500 µg/liter for the scud Gammarus pseudolimnaeus. Acute values for fish range from 3.9 µg/liter for the fathead minnow in soft water to 280 µg/liter for rainbow trout in hard water. In fresh water, the acute toxicity of silver appears to decrease as hardness increases. Soluble compounds, such as silver nitrate, are generally much more toxic than insoluble compounds. Chronic values ranging from 2.6 to 29 µg/liter are reported for Daphnia magna. Two early life stage studies with rainbow trout report chronic values of 0.12 µg/liter. Acute-chronic ratios for Daphnia magna and rainbow trout are 2.0 and 54, respectively. Fresh water aquatic plants appear to be more resistant to silver than the more sensitive animals.

Acute values for saltwater organisms range from 4.7 µg/liter for the summer flounder to 1,400 µg/liter for the sheepshead minnow. A chronic value of 18 µg/liter, and an acute-chronic ratio of 14 is reported for the mysid shrimp.

Reduced cell numbers are observed in the saltwater alga Skeletonema costatum after exposure to 130 µg/liter of silver.

Excess silver can induce selenium, vitamin E, and copper deficiency symptoms in animals fed adequate diets, and can aggravate deficiency symptoms in animals whose diets lack one or more of these nutrients. These effects are reported in dogs, sheep, pigs, chicks, turkey poults, and ducklings.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: $e^{(1.72 [\ln(\text{hardness})] - 6.52)}$ µg/liter
Chronic toxicity: No criteria have been established

Saltwater

Acute toxicity: 2.3 µg/liter
Chronic toxicity: No criteria have been established

Human Health

Criterion: 50 µg/liter

Primary Drinking Water Standard: 50 µg/liter

OSHA Standard: 10 µg/m³ TWA

ACGIH Threshold Limit Values: 0.1 mg/m³ (metal)
0.01 mg/m³ (soluble compounds)

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- DOULL, J., KLAASSEN, C.D., and AMDUR, M.O. 1980. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd ed. Macmillan Publishing Co., New York. 778 pages
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials. 4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Silver. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-071
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

ZINC

Summary

Ingestion of excessive amounts of zinc can cause fever, vomiting, and stomach cramps. Zinc oxide fumes can cause metal fume fever. Inhalation of mists or fumes may irritate the respiratory tract, and contact with zinc chloride may irritate the eyes and skin. High levels of zinc in the diet have been shown to retard growth and produce defective mineralization of bone.

Background Information

Zinc generally exists in nature as a salt with a valence of +2, although it is also found in four other stable valences.

CAS Number: 7440-66-6

Chemical Formula: Zn

IUPAC Name: Zinc

Chemical and Physical Properties

Atomic Weight: 65.38

Boiling Point: 907°C

Melting Point: 419.58°C

Specific Gravity: 7.133 at 25°C

Solubility in Water: Insoluble; some salts are soluble

Solubility in Organics: Soluble in acid and alkali

Vapor Pressure: 1 mm Hg at 487°C

Transport and Fate

Zinc can occur in both suspended and dissolved forms. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. Suspended (undissolved) zinc may be dissolved following minor changes in water chemistry or may be sorbed to suspended matter. The predominant fate of zinc

Zinc

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in aerobic aquatic systems is sorption of the divalent cation by hydrous iron and manganese oxides, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their compositions and concentrations; the pH and salinity of the water; the concentrations of complexing ligands; and the concentration of zinc. Concentrations of zinc in suspended and bed sediments always exceed concentrations in ambient water. In reducing environments, precipitation of zinc sulfide limits the mobility of zinc. However, under aerobic conditions, precipitation of zinc compounds is probably important only where zinc is present in high concentrations. Zinc tends to be more readily sorbed at higher pH than lower pH and tends to be desorbed from sediments as salinity increases. Compounds of zinc with the common ligands of surface waters are soluble in most neutral and acidic solutions, so that zinc is readily transported in most unpolluted, relatively organic-free waters.

The relative mobility of zinc in soil is determined by the same factors affecting its transport in aquatic systems. Atmospheric transport of zinc is also possible. However, except near sources such as smelters, zinc concentrations in air are relatively low and fairly constant.

Since it is an essential nutrient, zinc is strongly bioaccumulated even in the absence of abnormally high ambient concentrations. Zinc does not appear to be biomagnified. Although zinc is actively bioaccumulated in aquatic systems, the biota appear to represent a relatively minor sink compared to the sediments. Zinc is one of the most important metals in biological systems. Since it is actively bioaccumulated, the environmental concentrations of zinc probably exhibit seasonal fluctuations.

Health Effects

Testicular tumors have been produced in rats and chickens when zinc salts are injected intratesticularly, but not when other routes of administration are used. Zinc may be indirectly important with regard to cancer since its presence seems to be necessary for the growth of tumors. Laboratory studies suggest that although zinc-deficient animals may be more susceptible to chemical induction of cancer, tumor growth is slower in these animals. There is no evidence that zinc deficiency has any etiological role in human cancer. There are no data available to suggest that zinc is mutagenic or teratogenic in animals or humans.

Zinc is an essential trace element that is involved in enzyme functions, protein synthesis, and carbohydrate metabolism. Ingestion of excessive amounts of zinc may cause fever, vomiting,

stomach cramps, and diarrhea. Fumes of freshly formed zinc oxide can penetrate deep into the alveoli and cause metal fume fever. Zinc oxide dust does not produce this disorder. Contact with zinc chloride can cause skin and eye irritation. Inhalation of mists or fumes may irritate the respiratory and gastrointestinal tracts. Zinc in excess of 0.25% in the diet of rats causes growth retardation, hypochromic anemia, and defective mineralization of bone. No zinc toxicity is observed at dietary levels below 0.25%.

Studies with animals and humans indicate that metabolic changes may occur due to the interaction of zinc and other metals in the diet. Exposure to cadmium can cause changes in the distribution of zinc, with increases in the liver and kidneys, organs where cadmium also accumulates. Excessive intake of zinc may cause copper deficiencies and result in anemia. Interaction of zinc with iron or lead may also lead to changes that are not produced when the metals are ingested individually.

Toxicity to Wildlife and Domestic Animals

Zinc produces acute toxicity in freshwater organisms over a range of concentrations from 90 to 58,100 µg/liter and appears to be less toxic in harder water. Acute toxicity is similar for freshwater fish and invertebrates. Chronic toxicity values range from 47 to 852 µg/liter and appear to be relatively unaffected by hardness. A final acute-chronic ratio for freshwater species of 3.0 has been reported. Although most freshwater plants appear to be insensitive to zinc, one species, the alga Selenastrum capricornutum, exhibited toxic effects at concentrations from 30 to 700 µg/liter. Reported acute toxicity values range from 2,730 to 83,000 µg/liter for saltwater fish and from 166 to 55,000 µg/liter for invertebrate saltwater species. Zinc produces chronic toxicity in the mysid shrimp at 166 µg/liter. The final acute-chronic ratio for saltwater species is 3.0. Toxic effects are observed in saltwater plant species at zinc concentrations of 50 to 25,000 µg/liter. Bio-concentration factors of edible portions of aquatic organisms range from 43 for the soft-shell clam to 16,700 for the oyster.

Zinc poisoning has occurred in cattle. In one outbreak, poisoning was caused by food accidentally contaminated with zinc at a concentration of 20 g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited severe enteritis, and some died or had to be slaughtered. Postmortem findings showed severe pulmonary emphysema with changes in the myocardium, kidneys, and liver. Zinc concentrations in the liver were extremely high. Based on relatively limited data, some researchers have speculated that exposure to excessive amounts of zinc may

constitute a hazard to horses. Laboratory studies and findings in foals living near lead-zinc smelters suggest that excessive exposure to zinc may produce bone changes, joint afflictions, and lameness. In pigs given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/kg, deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints. High concentrations of zinc were found in the liver.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: $e^{(0.83[\ln(\text{hardness})] + 1.95)}$ $\mu\text{g/liter}$
Chronic toxicity: 47 $\mu\text{g/liter}$

Saltwater

Acute toxicity: 170 $\mu\text{g/liter}$
Chronic toxicity: 58 $\mu\text{g/liter}$

Human Health

Organoleptic criterion: 5 mg/liter

Secondary Drinking Water Standard: 5 mg/liter

NIOSH Recommended Standard: 5 mg/m^3 (zinc oxide)

OSHA Standard: 5 mg/m^3 TWA (zinc oxide)

ACGIH Threshold Limit Values:

Zinc chloride fume:	1 mg/m^3 TWA
	2 mg/m^3 STEL
Zinc oxide fume:	5 mg/m^3 TWA
	10 mg/m^3 STEL
Zinc oxide dust:	10 mg/m^3 TWA (nuisance particulate)
Zinc stearate:	10 mg/m^3 TWA (nuisance particulate)
	20 mg/m^3 STEL

REFERENCES

- AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, Ohio. 488 pages
- CASARETT, L.J., and DOULL, J., eds. 1975. Toxicology: The Basic Science of Poisons. Macmillan Publishing Co., New York. 768 pages
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances. Data Base. Washington, D.C. October 1983
- SAX, N.I. 1975. Dangerous Properties of Industrial Materials. 4th ed. Van Nostrand Reinhold Co., New York. 1,258 pages
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1980. Ambient Water Quality Criteria for Zinc. Office of Water Quality and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-079
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1984. Health Effects Assessment for Zinc. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-HO48
- U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1985. Health Assessment Document for Dichloromethane (Methylene Chloride). Office of Health and Environmental Assessment. Washington, D.C. February 1985. EPA 600/8-82/004F
- WEAST, R.E., ed. 1981. Handbook of Chemistry and Physics. 62nd ed. CRC Press, Cleveland, Ohio. 2,332 pages

DRAFT

Hazardous Substance

CERCLA Designation

Barium	
Cadmium	CWA§307a
Chromium	CWA§307a
Copper	CWA§307a
Lead	CWA§307a
Nickel	CWA§307a
Mercury	CWA§307a, CAA§112, RCRA§3001
Silver	CWA§307a
Zinc	CWA§307a
Cyamide	CWA§307a
1,1,1-Trichloroethane	CWA§307a, RCRA§3001
Tetrachloroethylene	CWA§307a, RCRA§3001
Trichloroethylene	CWA§311b4, 307a, RCRA§3001
1,1,Dichloroethylene	CWA§307a, RCRA§3001
Trichloroflouromethane	RCRA§3001
Chloroform	CWA§311b4, 307a, RCRA§3001
Phenanthrene	CWA§307a
Benzene	CWA-Section 311b4, 307a, CAA-Sect.112 RCRA§3001
Toluene	CWA§311b4, 307a, RCRA§3001
Pyrene	CWA§307a
PCBs	CWA§311b4, RCRA§3001

9/80
Quanta

Hazardous
Substance

CERCLA
Designation

Pls. typewrite
draft. Randye

Barium - TAP ~~RCRA § 307a~~
Cd. - CWA § 307a
Cr - CWA § 307a
Copper - CWA § 307a
Lead - CWA § 307a
Nickel - CWA § 307a
Mercury - CWA § 307a, CAA-§ 112, RCRA § 3001
Silver - CWA § 307a
Zinc - CWA 307a
Cyanide - CWA § 307a

1,1-Trichloroethane - CWA-307a, RCRA § 3001

Tetrachloroethylene - CWA-307a, RCRA § 3001

Trichloroethylene - CWA-311b4, 307a, RCRA § 3001

~~Dichloromethane~~ -

1,1, Dichloroethylene - CWA § 307a, RCRA § 3001

Trichlorofluoromethane - RCRA § 3001

Chloroform - CWA-§ 311b4, 307a, RCRA § 3001

Phenanthrene - CWA-§ 307a

Benzene - CWA - Section 311b4, 307a, CAA - Sect. 112, RCRA § 3001

Toluene - CWA-§ 311b4, 307a, RCRA § 3001

Pyrene - CWA § 307a,

PCB - CWA-§ 311b4, RCRA 3001